VOL. I NOVEMBER, 1932 NO. 5

THE JOURNAL OF PEDIATRICS

A MONTHLY JOURNAL DEVOTED TO THE PROBLEMS AND DISEASES OF INFANCY AND CHILDHOOD

Official Organ for
THE AMERICAN ACADEMY OF PEDIATRICS

EDITORS

BORDEN S. VEEDER
St. Louis

HUGH McCULLOCH
St. Louis

EDITORIAL BOARD

KENNETH D. BLACKFAN, Boston
THOMAS B. COOLEY, Detroit
HAROLD K. FABER, San Francisco
ROBERT A. STRONG, New Orleans
PHILIP VAN INGEN, New York
CLIFFORD G. GRULEE, Chicago
(Ex officio)

Published by THE C. V. MOSBY COMPANY, 3523-25 Pine Blvd., Saint Louis, U. S. A.

Entered at the Post Office at St. Louis, Mo., as Second Class Matter
Copyright 1932 by The C. V. Mosby Co.

FOR ETHICAL PRACTITIONERS ONLY

Compare these cost figures with those you now pay

$5,000.00 Accidental Death — $30.00 PER YEAR, $25 weekly indemnity, health and accident
$10,000.00 Accidental Death — $60.00 PER YEAR, $50 weekly indemnity, health and accident
$15,000.00 Accidental Death — $90.00 PER YEAR, $75 weekly indemnity, health and accident

Send for applications for membership

Physicians Casualty Assn.—Physicians Health Assn.
E. E. ELLIOTT, *Secretary-Treasurer*
301-13 City National Bank Bldg. OMAHA, NEBRASKA

How Much Sun Does the Infant Really Get? ◆

Not very much: (1) When the baby is bundled to protect against weather or (2) when shaded to protect against glare or (3) when the sun does not shine for days at a time. Mead's 10 D Cod Liver Oil with Viosterol offers protection against rickets 365¼ days in the year, in measurable potency and in controllable dosage. *Use the sun, too.*

Please enclose professional card when requesting samples of Mead Johnson products to cooperate in preventing their reaching unauthorized persons

The Journal of Pediatrics

VOL. 1 NOVEMBER, 1932 No. 5

Original Communications

CHILD HEALTH AND PROTECTION FROM THE DOCTOR'S VIEWPOINT

E. H. CARY, M.D.

DALLAS, TEXAS.

President of the American Medical Association

WHEN honored by an invitation to be a guest of the Iowa White House Conference on Child Health and Protection, I am frank to say that I did not fully realize the far-reaching influence such a gathering would probably have upon the future attitude of the members of our profession and the public.

It has been said that the medical profession can be criticized for failing to carry to the people the knowledge which it possesses. As a profession, we have always had certain inhibitions—we have shunned publicity. Doctors have believed that the public would misinterpret any unusual effort on their part. From this assumption the medical profession has always turned, to avoid even the appearance of being unethically engaged.

There are many problems, some of them grave, connected with children, all of which interest the members of our profession. We should not shirk our responsibility. We should be willing to meet the layman who is interested in the welfare of the child, for the purpose of cooperation. The layman has been willing to seek knowledge; this knowledge can only be gained through the members of our profession. Leadership is associated with the tactful use of knowledge.

I am constrained to believe that the layman recognizes that leadership in health matters should be based upon a knowledge of the medical sciences, and further, that this knowledge should be fortified with a clinical experience, ripened through contact with suffering humanity.

So it behooves us as a profession to recognize these human attitudes and aptitudes, and assume that leadership in all matters relating to medicine, which is our natural sphere. Our assumption is based upon our education, training, and desire to serve.

Address delivered at the Iowa White House Conference, Des Moines, Iowa, April 14, 1932.

We must recognize that the medical man has a dual occupation, for in the practice of medicine the relationship between the doctor and the patient is a very personal one. The physician is respected for his knowledge, he often receives a friendly affection and is trusted with many confidences. This dependence can be beautiful in human relations.

Too, he is responsible in a civic way, for he must realize his obligation to take part in public medical service. He should interest himself in the activities of organizations which may be trying to meet certain unsolved health problems. Many of these have arisen chiefly from an awakened public health consciousness, which has come from the revelation of medical research.

The medical profession does not want to believe that the enlightened public desires to interfere with the private practice of medicine, but rather that it is groping for a more widespread application of what it believes to be medical discoveries. Since it is untrue that we wish to deny the public the benefit of all of the knowledge which would be helpful to it, our public relations should be so guided that we will not only join the layman, but willingly place our knowledge and leadership on the altar of Public Good. Should this be accomplished, I firmly believe that the people would welcome our cooperative efforts on their behalf.

We practitioners call each other colleagues. The public health officer must also be a colleague in the truest sense. Our public relations should be such that any program of salvage, whether from pain or death, would be one in which we should all share a common human interest.

In practicing curative medicine, we have no desire to subsist upon the ills of man which are preventable. We realize the ever-increasing breadth of preventive service and that our first task is to save the life of our fellow man. If we were very practical in our thinking, it might occur to us that it requires thought and labor to preserve and make efficient those who are saved.

As medical practitioners we are no longer isolated. We are definitely committed to preventive measures which deal with groups, and these if effective, seem to deprive the profession of individual practice.

It is our first duty to cooperate to the limit in preventing disease, regardless of how it affects us. This we can do in good conscience, for it is also true that the complexities of life create new possibilities for the medical man, and his usefulness is not lessened, but apparently ever enlarged.

There can be no contest in doing good; the service one renders in this life brings its compensations.

The people themselves have a conception that scientific medicine has gone far in its development, and through its application, it can limit unnecessary illnesses. They have been wondering whether the present

public health organization is adequately trained or if it needs to be enlarged with many more expertly trained men to provide maximum care.

We have it in our power to guide all these questionings and efforts to advance preventive medicine. The profession should be the major force in proposals for solution of the vast problems concerning adequate medical service.

Social changes are undoubtedly confronting us. We have had a tremendous number presented already. Social trend should be studied as medicine is a social function, and all of these changes in some way affect the practice of medicine.

There is a good reason for believing that one of the greatest factors in human happiness, and I think in civilization, is the preservation of, and continued flexible growth of the medical profession to meet human needs. We must not be deterred from contributing our weight of opinion and battling for leadership in any lay organization or foundation interested in health matters, which seems to have public support. Rather should we more definitely express our convictions and cooperate with the people, recognizing that medicine has assumed a public character.

It is unfortunate that every doctor is not civic-minded, for a well developed civic instinct would enlarge his point of view.

Where public welfare laws represent the true interest of the people, the individual relation of the doctor to his patient is not disturbed. Every county medical society should be in touch with new conditions and within the society there should be an intelligent plan devised to prevent economic disturbance of the practice of medicine. A few of the wisest members of the county society should deal with such matters as they arise, and all differences of opinion should be settled.

The spirit of cooperation can become an economic asset, if the medical profession and the public have the same objective—it insures the improvement of the health and happiness of the people.

What I have said has a wide application, although I am here to discuss more particularly child health, but it is all interrelated.

I believe that we will all agree concerning the modern view of prenatal care, but this part of a child's life, and in all probability its first two or three years, would lack interest to you on this occasion. Yet it is impossible to speak of Child Health and Prevention of Diseases from the Doctor's Point of View without a searching history of the forbears of the child. Heredity is a marvelous thing. When the little patient inherits that something which is represented in vim, vigor and vitality, the doctor has an added advantage. He finds it easy to steer such an infant over the rough waters of babyhood. Even his directions may not always be followed, but his results may be so good he is generously

appreciated. On the other hand, the physical qualities of the child lacking a sturdy stamina of inheritance puts the doctor on the defensive, with losses here and there, even though there may have been the most meticulous care.

Undoubtedly the highly trained pediatrician who was primarily an educated and experienced general physician, is the most desirable doctor for children. He bridges what was a chasm of despair for many well-meaning mothers and family doctors who really could not fathom the needs of the infant.

The efficient pediatrician is making a most excellent demonstration of preventive medicine, in which the people are being educated in the wisdom of securing medical advice before disease is manifested. The unsafe pediatrician is cursed with a self-assurance which leads him to make a diagnosis of special conditions without comprehensive training in special fields. The spirit of cooperation here is greatly needed. Able medical men should be willing to lend their knowledge to each other for the purpose of making a correct diagnosis.

As I understand it, a pediatrician's point of view comprehends a physically fit child to be developed with a proper diet of quality and quantity. He should have a knowledge of the organic troubles which might arise, due to infections in various places such as the sinuses, tonsils, and gastrointestinal tract, as well as recognizing infectious diseases of a general nature.

The mental health of the child is often closely associated with its social relations. Family adjustments are needed and are brought about through a correct use of the accumulated knowledge related to this subject. There are environmental situations too, which are difficult to control. These are of great importance to the child, requiring judgment and tact on the part of the physician to harmonize them.

I would say that the wise pediatrician is here to stay. He is needed and has proved his worth. He it is who can most readily recognize that train of unfortunate circumstances in a child's life which has brought stress beyond the physical and mental capacity of its nervous system.

We are beginning more and more to sense the fact that future civilization lies with the proper development of the child. Childhood has never had in all the years gone by, such fortunate opportunities for health and happiness. Every facility is offered for a more perfect development of our children in this era. It lies within the province of the medical profession to direct the family and child so that cooperation will be the result. In recognition of the opportunities for education of the family, the pediatrician should have the encouragement of the profession. Man is but a grown-up child, health standards and increased efficiency are more likely to be obtained, if we are permitted to direct the health of the child.

We must recognize that each individual must, if possible, become a self-sustained unit. His physical defects must be eliminated, and if his nervous system is normal, his capacity for self-support, comfort, and happiness, should be realized.

This cannot be accomplished without danger, except by intelligent application of modern medical information. There can be no separation of the psychologists, psychiatrists, social workers, and specialists, in these various medical fields. If this is not duly appreciated and the needs of cooperation and consultation made readily available, there will be chaos.

Unification of control in public health matters cannot be divided without great loss to the nation. Dictation or direction is not needed from bureaus separated from the public health agencies.

Iowa is a pioneer state in its work for children. The citizens of other states have been interested, and have watched with increasing approval what has been accomplished. This work has undoubtedly had a far-reaching influence upon the public health service and child welfare departments throughout the country. Most interesting is your child welfare research station—and with such a demonstration the people of this country should rest assured that the good accomplished will be of lasting benefit to future generations.

At the last White House Conference, it was reported that of forty-five million children, thirty-five million are reasonably normal, six million are improperly nourished, one million have defective speech, and one million have weak or defective hearts, six hundred and seventy-five thousand present behavior problems, four hundred and fifty thousand are mentally retarded, three hundred and eighty-two thousand are tuberculous, three hundred and forty-two thousand have impaired hearing. eighteen thousand are totally deaf, three hundred thousand are crippled, fifty thousand are partially blind, fourteen thousand are totally blind, two hundred thousand are delinquent, five hundred thousand are dependent. Of these ten million deficients it is said that eighty per cent are not receiving the necessary attention. This is a challenging statement made in a high place. It is probably true. It is imperative that the medical profession lead those forces of society which are willing to help correct and decrease the number of abnormals so far as it is humanly possible to do so.

We can boldly take sides and lead those forces which demand for the child the application of certain suggestions brought out at the last White House Conference, and so succinctly summarized by our distinguished Secretary of the Interior, Dr. Ray Lyman Wilbur. He lays stress upon social factors for the general good of the child to be had through education of the parent and school-teacher.

He pleads that the adult should understand the child, and that the child be not required to do all the comprehending. Every mother should have prenatal and postnatal care.

The public should recognize that the child must be periodically examined; its physical forces known; proper advice given, if young manhood is to gain maximum efficiency. This examination should be all-inclusive and repair work suggested.

This era carries a burden of untold accidents, producing cripples, often death. Vast numbers can be saved if our educational forces cooperate in a more united and impressive effort toward riveting the child mind upon traumatic possibilities.

Our health forces, both public and private, should guard the child from communicable disease to which it might be exposed by contact with those infected at home or school. Food requirements too, should be properly worked out.

Hygiene, which is so well understood, should be practiced by the growing youth of the land.

The principle that one is born free is generally accepted. Every child, however, has an individuality which should be observed and guided and its dominant qualities developed, with the hope that the state will prosper when there are fewer square pegs in round holes.

Reverence and faith should be cultivated. Certainly one's home life should be surrounded with those high qualities which lead to faith, hope and charity. It has been said that Young America lacks reverence. Maybe it does. If the truth is magnified, hypocrisy discouraged, maybe there would be a better understanding. Moral concepts differ under different conditions, but there are a few things which remain eternal.

We doctors believe in the prevention of disease; in prevention of accidents; in prevention of losses from every cause. We believe in treatment when disease occurs. This may contemplate any method best adapted to the case in hand.

Medicine is broad in concept and application. It is at the service of mankind. Those who lead in the great movement of understanding between the people and the profession must continuously endeavor to focus public attention upon its blessings, sporadic effort is not enough. Action has to be sustained till it becomes automatic for the best result from mass endeavor.

Preventive medicine concerned with the growth and development of childhood discloses information, which can be made to bring into being the well-born child—to further see to it that he thrives lustily without halt during infancy; that he develops physically, mentally and socially, acquiring a sound education with which he can pass through adolescence into a more secure maturity.

Continuity of the scientific advance in the cause of prevention of disease, is a long story. It was made possible by the slow evolution of valuable scientific instruments which was extremely *slow* in being developed to where the minute enemies of man could be identified and disclosed to the profession of medical men. The study of infectious diseases became profitable within our era and today we stand upon a high point, where we may look forward or turn and point to the possibilities of the future, realizing how we have yet to decrease the death rate of children under five, who constitute four-fifths of the mortality of all who die from infectious diseases; and the further fact that from birth to the tender age of five, twenty-five per cent of all deaths occur.

At this point, not to mention what occurs from the age of five to maturity, we have yet a task of education, and more education to change the record—by driving back the forces of the grim reaper of youthful and tender lives.

Someone has said that "The child is a dynamic personality in the making. It is a composite individuality with evanescent manifestations of growth and development. It is a veritable little laboratory, transforming food into growth, experience into life habits, environment into personality," all of which is true.

We must guard the normal growth of the mind and body of the children of our land. As the future of our country is interwoven in this great bundle of life, we should more and more help direct the destiny of this fair land.

Our knowledge is increasing—our desire to help is evident. From meetings of this character a new spirit of conquest is born. Let it guide aright the hearts of all, so that *cooperation between the layman and doctor is made a reality.*

PARATHYROIDECTOMY IN GENERALIZED OSTEITIS FIBROSA CYSTICA

REPORT OF A CASE IN A CHILD TWO AND ONE-HALF YEARS OF AGE

JOHN FITCH LANDON, M.D.
NEW YORK, N. Y.

OSTEITIS fibrosa cystica is a condition characterized by the rarefaction in localized areas of the bone tissue and its substitution by fibrous tissue or by a cyst. Because of the similarity of the pathologic changes in the skeleton in this disease and in osteomalacia, osteogenesis imperfecta and rickets, some authors have been led to regard all of these conditions as a disease entity with varying degrees of intensity, and with different osseous distribution. This view is probably erroneous, for the essential disturbance in rickets and in osteomalacia seems to be a laying down of bone of a type deficient in lime salts, commonly known as osteoid tissue; while osteoid tissue is not much increased if at all in osteitis fibrosa cystica, as has been well emphasized by Turnbull.[1] Von Recklinghausen[2] originally pointed out that there is metaplasia of bone tissue in this disease.

The first hint that there might be some relationship between the parathyroids and calcium metabolism came from experiments by Erdheim[3] who showed that the removal of parathyroids in rats was associated with lack of calcification of their teeth. Several years later, in 1907, he further commented on the occurrence of hypertrophy of the parathyroids in osteomalacia as observed at autopsy. He believed that this hypertrophy represented an inadequate attempt at compensation; in short, that it was the result and not the cause of the bone deficiency. Shortly after, Hoffeinz[4] reviewed 45 autopsies in which hypertrophy or disease of the parathyroids was found and discovered among them 27 cases of disease of the skeleton. Eighteen of these were osteitis fibrosa cystica and eight osteomalacia. In 1929 Bauer, Albright and Aub[5] reviewed the literature to date. Of 85 cases of bone disorder associated with abnormal parathyroids, 14 showed parathyroid adenomata, and the rest parathyroid hyperplasia. In the latter group there were 21 cases of osteomalacia, 15 of osteitis fibrosa cystica, and 3 of rickets. Mandl,[6] in 1925, was the first to try clinically to prove the relationship between the parathyroids and the bone disorders. Acting on the Erdheim assumption that in cases of osteomalacia an hypertrophy of the parathyroids represents an inadequate compensatory mechanism, he implanted the parathyroids from a patient who had just died into the abdominal wall of a patient thirty-eight years old who had been suffer-

Presented at a meeting of the Pediatrics Section of the New York Academy of Medicine, May 12, 1932.

ing from an osteitis fibrosa cystica and had been completely disabled for five years. Contrary to expectations, the patient became much worse. He then proceeded to remove a left parathyroid adenoma in the same patient, and the man, who previously had been unable to lift his legs from the bed, rapidly regained his strength and within three months was walking about with a cane. The obvious conclusion from

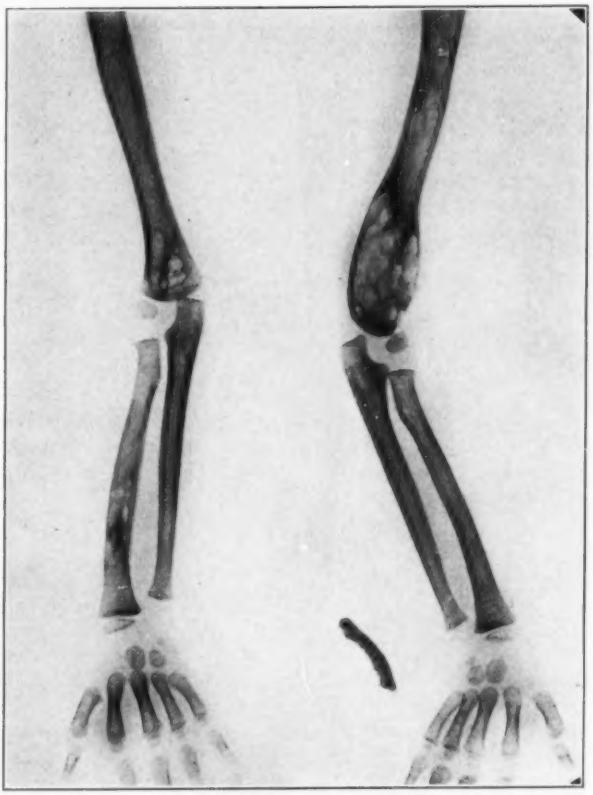

Fig. 1.—Before operation.

this case was that the parathyroids are primarily hypertrophied in osteitis fibrosa cystica, and probably secondarily hypertrophied in osteomalacia, as Erdheim suggested.

In 1930 Hunter,[7] in the Second Goulstonian Lecture, collected 14 cases of osteitis fibrosa cystica associated with parathyroid disturbance. In nine of these cases a parathyroid tumor was removed. Two cases in which normal parathyroid tissue was excised were but slightly improved. In the remaining three cases there was no operation on the

neck. Latent tetany was common after operation, but in only one case, that in which two parathyroid tumors were removed, was the tetany fatal. Polyuria was present in three cases, and in one instance bilateral renal calculi were discovered.[8] Recently Quick and Hunsberger[9] have reported a case in a twenty-six-year-old man, a parathyroid tumor being removed with subsequent marked improvement.

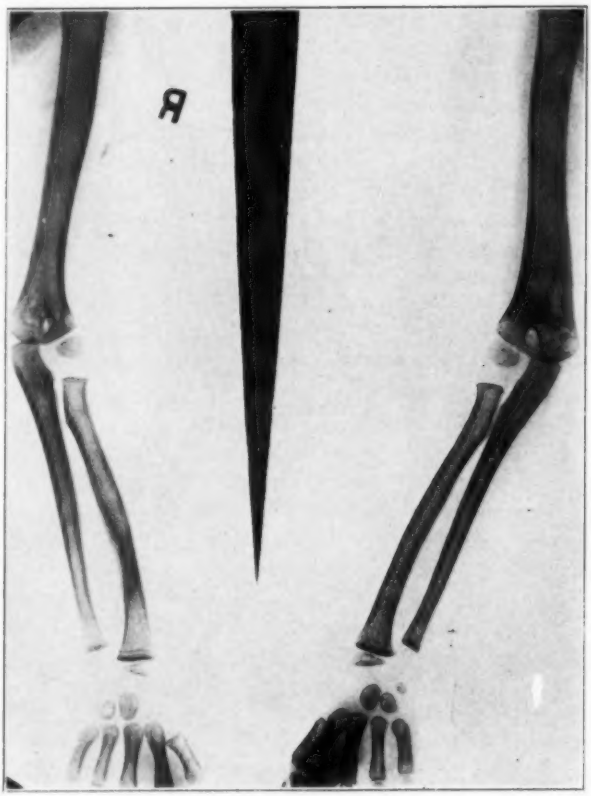

Fig. 2.—Three months after operation.

The disease occurs twice as often in the female as in the male. It is most common between the ages of thirty and fifty-five years. The youngest case reported to date is that by Amberg[10] in discussion of a paper by Ball at The Mayo Clinic. The patient was a fourteen-year-old girl. There have been no reports of parathyroidectomy in children. The case presented below is by far the youngest yet reported, and the first in which a parathyroidectomy has been attempted in a child.

CASE REPORT

J. H., white male, aged two and a half years, was admitted to the Roosevelt Hospital, service of Dr. Philip Van Ingen, on December 25, 1930, with a chief complaint of fever, cough, and irritability of twelve hours' duration. The patient was the only child of healthy parents, with a normal birth history. He was breast fed for two months. At six months of age, he was treated at Bellevue Hospital for a swelling of the left jaw. An incision was made, apparently for the purpose of obtaining tissue for study. At eleven months, he was admitted to St. John's Hospital, Brook-

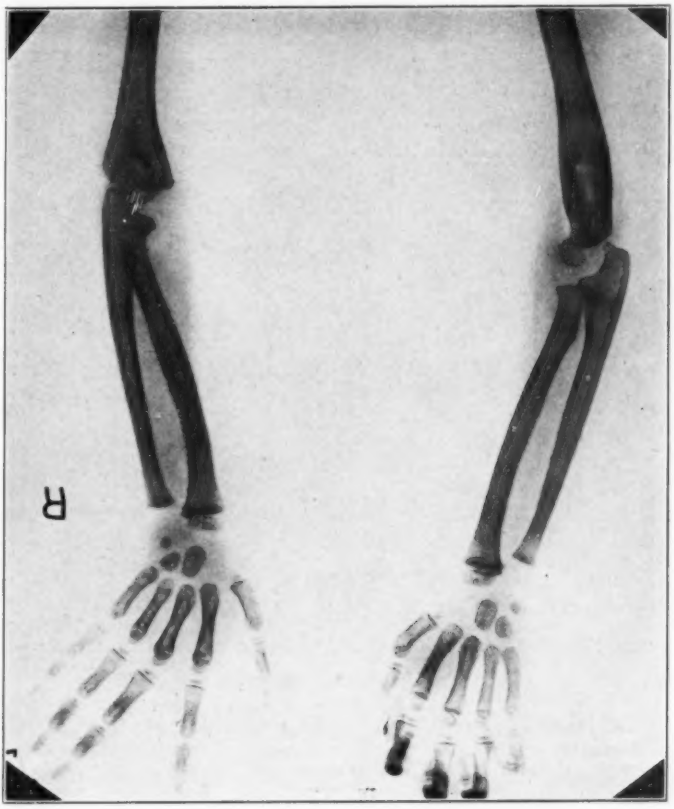

Fig. 3.—Six months after operation.

lyn, for a swelling over the left side of the head. Incision and drainage were again instituted. Several series of x-ray pictures of the bones were taken at this time, but the result was unknown to the mother. For the past few months it had been noticed that the child disliked being handled and limped slightly.

Physical examination revealed a fairly well-developed, poorly nourished, and rather pale white boy, not appearing acutely ill. Over the left frontoparietal region there was a shallow depression in the bone of about one inch in diameter with an overlying short scar. Over the inner condyle of the left humerus and extending nearly an inch above, there was a firm, somewhat elastic, immobile swell-

ing, apparently subperiosteal. The patient noticeably objected to deep pressure over any of the long bones. The left leg was slightly shorter than the right. Height was 35 inches. Tonsils were hypertrophied and diseased and there was a general glandular enlargement. The teeth were markedly carious. Otherwise abnormal physical findings were absent. Roentgenograph of the whole skeletal system showed what appeared to be a cystic degenerative process in the following bones: skull, left and right humeri, right radius, both heads of humeri, pelvis, both femora, both tibiae and right astragulus. The cortex of the bone appeared intact except in the lower right tibia, where the medial side of the tibia was broken through. Blood count: Hemoglobin, 69 per cent; R.B.C., 4,200,000; W.B.C.,

Fig. 4.—Before operation.

6,400; polymorphonuclear cells, 68 per cent; lymphocytes, 32 per cent. Mantoux and Schick tests were negative. Urine was negative. Wassermann reaction was negative. Diagnosis: Osteitis fibrosa cystica; acute tonsillitis; generalized glandular enlargement; dental caries.

Estimations of the serum calcium and phosphorus over the next three months were as follows:

Jan. 20	Calcium 10.5	Phosphorus 3.03
Feb. 2	Calcium 13.9	Phosphorus 2.8
Feb. 11	Calcium 13.5	Phosphorus 2.15
Mar. 31	Calcium 19.4	Phosphorus 5.5

No attempt was made to determine the calcium balance because of incontinence of the child.

Viosterol, 250 D., was given in doses ranging from five drops twice a day, to ten drops three times a day, without any demonstrable change in the child's con-

dition. Yellow phosphorus, grains $\frac{1}{100}$, twice a day, was administered for a short period without apparent effect.

In the middle of March, 1931, a small mass was palpated near the left thyroid region. Because of the hope that this might be a parathyroid adenoma, it was decided to explore the thyroid region and attempt to remove the parathyroid tissue. The operation was performed on April 1 by Dr. James Russell and Dr. Howard Patterson. The description of the operative procedure is as follows:

"The posterior surface of each lateral lobe was carefully inspected, following the usual methods of exposing the thyroid gland. On the posterior surface of the left lateral lobe, lying in the capsule, was a small oval brownish mass which was thought to be parathyroid tissue. This was removed and several small bits of tissue

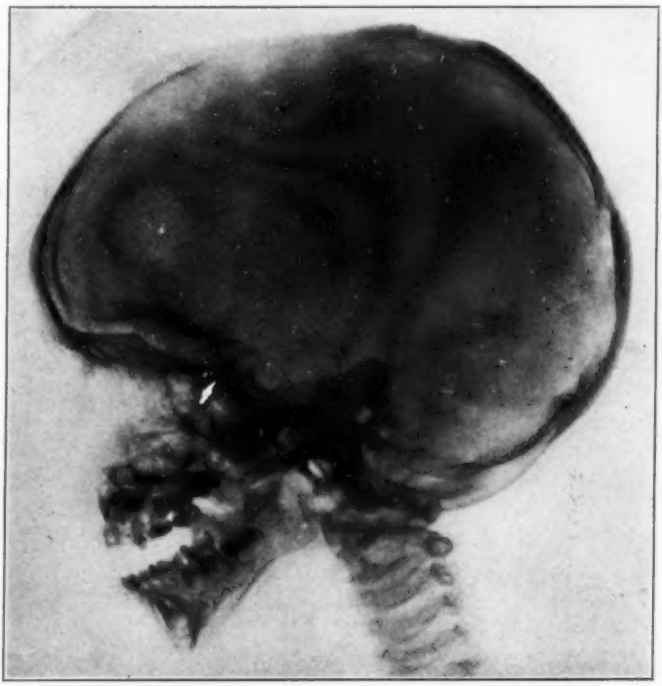

Fig. 5.—Six months after operation.

were removed from the posterior aspect of each lateral lobe. A small, firm lymph node was then removed from the right inguinal region for pathologic examination." The postoperative course was uneventful. There was no evidence of tetany at any time.

The pathologic report of the five sections removed is as follows: "The largest piece is a lymph node which shows normal structure with slight interstitial fibrosis and much swelling and multiplication of reticular cells. The piece from the right side has a glandular structure with considerable fibrous tissue between and at the periphery. There are small clusters of round cells, not lymphocytes. Other cells are clear, like the chief cells of the parathyroid. In the gland is some colloid. Of the pieces from the left side, one shows a compact cellular structure, the solid

strands of small cells. There is a delicate capsule and there are numerous capillaries. A second piece is fat. A third piece is mostly fat, but at one side is a structure showing clear cells and smaller, pale round nuclei. Diagnosis: Portions of parathyroid, 3; fat, 1; hyperplastic lymph node, 1."

By the first of May there was noted marked general improvement in the boy's condition. His irritability and dislike of being touched had disappeared, and he

Fig. 6.—Before operation.

was walking and running about the ward without any apparent limp. His height was now 37 inches. X-ray reports were as follows:

April 20: "Films of the long bones of the arms and legs demonstrate considerable improvement in cystic degenerative areas as compared with previous examination."

April 29: "Films of the lower extremities demonstrate what appears to be slight improvement in the cystic degenerative areas of the femur and tibia of both sides."

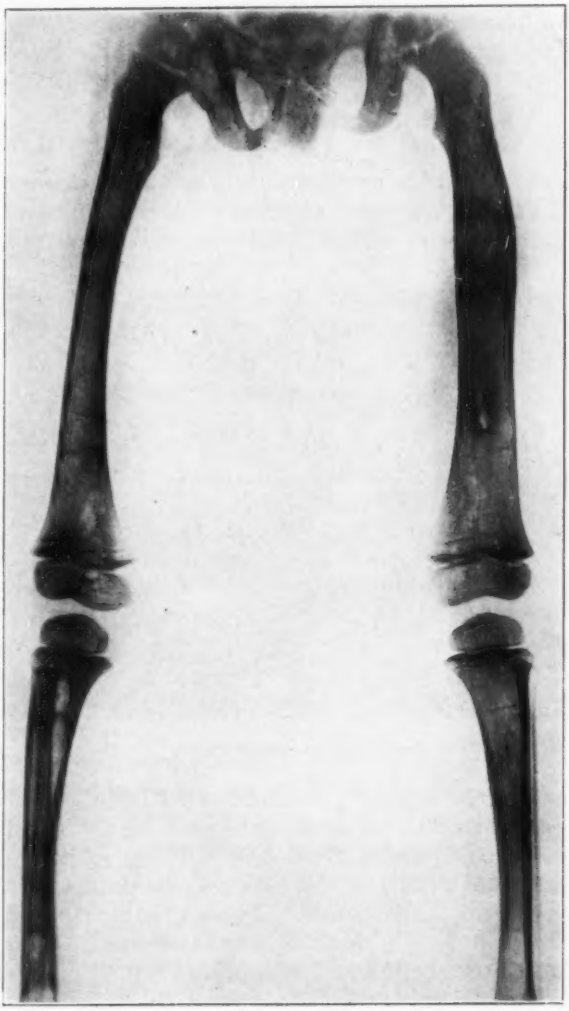

Fig. 7.—Three months after operation.

May 9: "Films of the long bones in comparison with previous examinations demonstrate marked improvement in cystic bone degenerative areas."

July 24: "Films particularly of femora show apparent signs of repair with increase in lime salt deposits in the bones."

On April 20 blood chemistry showed calcium 13 and phosphorus 5.

The patient was readmitted October 6, 1931, complaining of a lump on the head of one month's duration, starting as a small protuberance and rapidly increasing in size. The swelling was approximately two inches in diameter, fluctuant and nontender. X-ray examination of the skull showed a large destructive area of bone in the left temporal region with questionable early destructive lesion in the left occipital also. The blood calcium at this time was 13.0 and the phosphorus 3.28. Six months subsequently, however, this swelling had entirely disappeared from the head, and the roentgenogram showed definite evidence of bony healing. During this period, the patient had received no treatment of any kind.

DISCUSSION

Osteitis fibrosa cystica or hyperparathyroidism is characterized by high blood calcium, low blood phosphate, a greatly increased plasma phosphatase, a negative calcium balance at the expense of the bone,

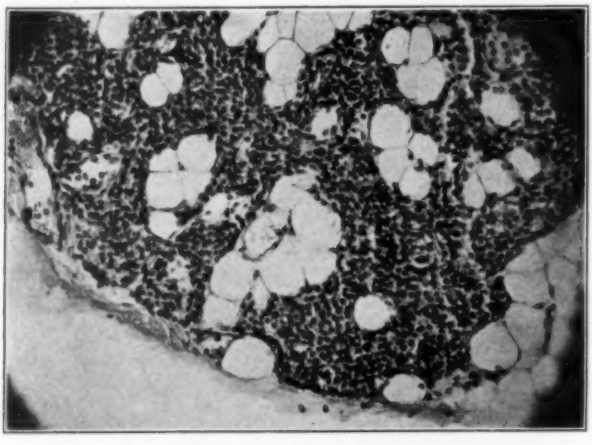

Fig. 8.—Section of parathyroid removed.

generalized muscular weakness, a lessened irritability of nerve, requiring in some cases three to four times the normal faradic stimulation for response, and vague pains in the skeleton. Radiographically there is a greatly diminished density of the bone with frequent cyst formation. Spontaneous fractures are common. The same picture can be duplicated experimentally in dogs by parathormone overdosage, except that, according to Collip,[11] the inorganic phosphorus reaches very high levels in the experimental animals but is always found to be normal or subnormal in spontaneous hyperparathyroidism in man.

It is most important to remember that the determination of the blood calcium and phosphate gives an indication of the height of the calcium stream but affords no information as to the direction of the stream, whether into the bone from the blood or out of the bone into the excretory organs; consequently it is necessary to determine the so-called

calcium balance which can only be done by careful estimation of the amount of calcium taken in in the food as compared with that eliminated in the feces and urine. Unfortunately due to incontinence of the child, this determination could not be made in our case.

Further, there are in addition to parathyroid activity a number of other factors which influence calcium metabolism. These include diet, acidosis, alkalosis, hyperthyroidism and vitamin D. Concerning the relationship between vitamin D and the parathyroid, Collip,[11] in a comprehensive review of the physiology of the parathyroid glands, states "that vitamin D hypercalcemia is definitely linked with increased calcium absorption appears to be well established. Whether it can cause mobilization of calcium from the bone reserves is not so clear; if it causes this latter effect, is the action a direct one, or do the parathyroid glands act as an intermediary? No final answer is to be had to this question with our present knowledge of the subject." There is considerable evidence to show that irradiated ergosterol acts through the intermediary of the parathyroid glands.

DuBois and his coworkers[12] have demonstrated that the normal urine-feces ratio of calcium excretion is inverted through the administration of parathormone. Normally 70 per cent to 90 per cent of calcium is excreted in the feces, but under parathormone treatment 10 per cent to 30 per cent is excreted in the feces and the rest in the urine. This may be taken as an indication of a more effective absorption from the intestine. Studies made by various observers on cases of osteitis fibrosa cystica have demonstrated that both urinary and fecal determinations of calcium and phosphate compare very closely to this experimental work.

In order to explain the abnormal excretory findings in these cases, Bauer, Albright and Aub[13, 14, 15] have evolved a theory that the difficulty is with the phosphate rather than with the calcium. In short, hyperparathyroidism causes increased elimination of phosphate, presumably endogenous, from the body as evidenced by a low serum phosphate. Since normally the body attempts to keep the calcium phosphate in a saturated solution in the blood, calcium is drawn off from the bone into the blood in order to preserve this saturation. This theory explains the high blood calcium, the increased urinary excretion of calcium (feces normally not affected), and the increased phosphate excretion. It is probable, according to these observers, on the basis of this theory, that the phosphate might prove of more aid therapeutically than calcium.

CONCLUSIONS

The most outstanding feature of the case here reported is the age of the patient. The literature contains no record of any case under the age of fourteen years. As pointed out by Albright,[16] it is of tremendous

importance to know whether the tumor of the jaw at the age of six months was an epulis. Unfortunately we have been unable to obtain this information from Bellevue Hospital. It is possible that the disease was present at six months. Further, the ends of the diaphyses show lines suggesting old rickets, but these lines may be part of the disease if present since birth.

The remarkable clinical and roentgenologic improvement following operation gave rise to the hope that a permanent cure had been effected. The rather sudden appearance of a cystic area in the right frontal region with an apparent meningocele with subsequent evidence of spontaneous healing leaves open the question of the specificity of the parathyroidectomy in this case. It is possible that an attempt to remove more parathyroid tissue will have to be made in the future.

The fact that the relatively high calcium and low phosphorus figures were not affected by the operation is in accord with the two cases quoted by Hunter,[7] in which normal parathyroid tissue was removed without change in the serum calcium and phosphorus, although there was not in either of these two cases any clinical or roentgenographic improvement.

REFERENCES

1. Turnbull: Brit. J. Surg. **19**: 203, 1931.
2. Von Recklinghausen, F.: Fortschr. Virchow, Berlin, 1891.
3. Erdheim: Mitt. a. d. Grenzgeb. d. Med. u. Chir. **16**: 632, 1906.
4. Hoffheinz: Arch. f. path. Anat. u. Physiol. **256**: 705, 1925.
5. Bauer, W., Albright, F., Aub, J. C.: J. Clin. Investigation **7**: 75, 1929.
6. Mandl, F.: Arch. f. klin. Chir. **143**: 245, 1926.
7. Hunter, Donald: Lancet **1**: 947, 1930.
8. Barr, Bulger, and Dixon: J. A. M. A. **92**: 951, 1929.
9. Quick, A. J., and Hunsberger, A.: J. A. M. A. **96**: 745, 1931.
10. Amberg: Proc. Staff Meeting, The Mayo Clinic **5**: 331, 1931.
11. Collip: Canadian M. A. J. **24**: 646, 1931.
12. Hannon, Shorr, McClellan, and DuBois: J. Clin. Investigation **8**: 215, 1930.
13. Albright, F., Bauer, W., and Aub, J. C.: J. Clin. Investigation **7**: 139, 1929.
14. Albright, F., and Ellsworth, R.: J. Clin. Investigation **7**: 183, 1929.
15. Bauer, W., Aub, J. C., and Albright, F.: J. Clin. Investigation **8**: 229, 1930.
16. Albright, F.: Personal communication.

2 East Fifty-Fourth Street.

IMMUNIZATION TO SCARLET FEVER BY THE INUNCTION METHOD

A Preliminary Report

Edgar E. Martmer, M.D.
Detroit, Michigan

WITH the announcement in 1924, by Drs. George and Gladys Dick, of the isolation of a toxin associated with the streptococcus of scarlet fever, interest in the control of scarlet fever was renewed. Since that time several methods of active immunization have been advocated. The Drs. Dick, utilizing the unmodified toxin, have been able to render groups Dick-negative by gradually increasing doses of toxin. This work has been duplicated by investigators throughout the world and is an accepted method of producing an active immunity to scarlet fever.

Several modifications of the dosage and time intervals as well as of the number of doses have been suggested by various investigators.

A combined toxin and vaccine partially detoxified by sodium ricinoleate has been suggested by Larsson and his group of investigators.

Scarlet fever toxin modified by formaldehyde according to the method of Ramon has been shown to produce immunity as demonstrated by a negative Dick reaction.

All of these methods are capable of rendering Dick-positive individuals Dick-negative after varying intervals of time. The percentage of successful immunization obtained by the different methods differs as well as the length of time between the administration of the immunizing agent and the appearance of a negative Dick reaction.

Despite the fact that all of the various methods of immunization to scarlet fever advocated have proved successful they have not been widely accepted because of the reactions accompanying their use.

There is a need for a method of immunization which is easily administered, painless or nearly so, and which does not cause either local or general reactions.

The method described here differs from those previously suggested in several respects. The work is based on skin absorption rather than subcutaneous or intramuscular injection of the scarlet fever toxin.

Lowenstein has reported the successful immunization to diphtheria by percutaneous administration of diphtheria toxin. Numerous investigators including Besredka, E. Nobel, A. H. Kegel and B. M. Gasul, J. Siegel and K. Hassman, A. F. Abt and B. F. Feingold, M. Stransky, R. Jakopp and A. Streit, and L. Lowry have used this method with success.

From the Children's Hospital of Michigan.
Submitted for publication, August 8, 1932.

Based on the success of this method in active immunization to diphtheria the investigation of scarlet fever was undertaken.

A review of the literature has not revealed any similar study.

METHOD OF PREPARATION OF MATERIAL

Dick toxin of a potency of 25,000 S. T. D. per cubic centimeter as standardized by the Michigan Department of Health was mixed with anhydrous lanolin so that one tube of ointment (2 cubic centimeters) contained 25,000 S. T. D. of toxin. The toxin used in making the ointment was freshly prepared and the finished ointment used within two months after its preparation. To overcome the odor of lanolin the base was perfumed.

METHOD OF ADMINISTRATION

The patient's back was washed with soap and water, dried thoroughly and then sponged with 70 per cent alcohol. After wiping off the excess alcohol, the back was allowed to dry before applying the material. The contents of one tube of ointment (25,000 S. T. D. of toxin) was applied to the cleansed surface and thoroughly massaged into the tissues by the hands. A rubber glove was worn by the nurse to facilitate the work and prevent any loss of material by absorption into her own hands.

Two groups of children were given inunctions of the scarlet fever ointment.

The first group consisted of two hundred and two (202) children (aged four to ten years), at the Convalescent Home of the Children's Hospital of Michigan at Farmington, Michigan. The entire group was Dick tested to determine which were Dick-positive and those who gave positive reactions were used in this series.

Ninety received five applications of ointment as outlined in the method of administration. The inunctions were given every second day, just before the children went to bed. The massaging consisted of rubbing the back ten times up and down, using as much pressure as possible so as to produce a slight degree of erythema in most cases. No soap or water was applied to the back for twenty-four hours. In this group the following results were obtained:

Total number treated (Dick-positive)	90
Total number Dick-negative (six months later)	42
Total number Dick-positive (six months later)	48

The second group of children was made up of a younger age group (two to six years) who were treated in the following manner:

Five applications of ointment were made with an interval of five days between applications. Following each application the back was massaged vigorously for five minutes.

There were 75 Dick-positive children in this group. At the end of six months, 61 of these were Dick-negative to one skin test dose of toxin. Four gave positive reactions and 10 were not available for testing.

Comparing the two groups it would appear that the longer interval (five days) and the more thorough massaging (five minutes in place of one-half to one minute) increases the number of negative reactions which develop following this method of immunization.

Combining the two groups the results, from the percutaneous administration of five doses of 25,000 units of scarlet fever toxin, were as follows:

155	Number of cases Dick-positive	
155	Number of cases treated	
103	Number of cases rendered Dick-negative	66%
52	Number of cases remaining Dick-positive (Six months following treatment)	34%

In the entire series no painful or severe reactions were observed. Local reactions at the site of the inunction developed in five cases. These consisted of a fine, papular eruption limited to the area. The rash usually appeared after the second or third dose and disappeared within thirty-six to forty-eight hours.

In one case, twenty-four hours after the third inunction, the child developed a generalized scarlatiniform rash which could not be distinguished from a true scarlet fever rash. It was practically limited to the trunk and no subjective symptoms were present. The temperature did not go above 99° F. by mouth, there was no headache, nausea or vomiting and no complaint of sore throat. The throat was not inflamed. The child had no known exposure to scarlet fever. The tongue did not follow the usual course observed in most cases of scarlet fever and the patient did not desquamate. A group of three Dick-positive children were intimately exposed to the child and they did not contract the disease.

It is possible this was an extremely mild case of scarlet fever but because of the symptoms and course it was felt to be a reaction to the application of the ointment.

It appears that scarlet fever toxin combined with anhydrous lanolin and applied percutaneously by massaging causes a Dick-positive reaction to become Dick-negative in a fairly large proportion of cases.

Further investigation of this method of active immunization to scarlet fever is being carried on because of the ease of administration, lack of local or general reactions; and the absence of the psychic factor involved in a series of hypodermic injections.

The writer wishes to express his indebtedness to Doctor Thomas B. Cooley for valuable advice and aid in preparation of this report.

749 David Whitney Building.

A PRACTICAL APPROACH TO THE MENTAL HEALTH OF CHILDHOOD WITH SPECIAL REFERENCE TO THE INTELLIGENCE ASPECTS

ESTHER L. RICHARDS, M.D.
BALTIMORE, MD.

THE mental health of childhood is a topic to which physicians have paid comparatively little attention. The behavior of childhood has been considered by the physician as well as the layman to belong in the province of ethics unless this behavior was so grossly out of the ordinary as to stamp the child as objectively abnormal. From a questionnaire sent during the White House Conference on Child Health and Protection to leading pediatricians throughout this country, it was learned that the term "mental health" was usually considered synonymous with the intellectual development of a child. One pediatrician wrote, "We are spending altogether too much time on feeble-minded children. I have always been able to diagnose them without the help of psychology. Nobody has been able to cure them." Our present generation of progressive medicine is coming to realize that it cannot dismiss the behavior of childhood in any such cursory manner. This point of view is well expressed by a young doctor who spent five or six years in preparing himself to practice pediatrics. A year after he had settled in a small city he wrote, "I have been greatly surprised to find that at least half of my practice takes me into the field of behavior difficulties in childhood that I do not know how to handle. Do you know of any medical school or hospital where I can get training along this line?" This inquiry is significant of the fact that it is to the physician that parents and teachers turn first of all for help in every matter pertaining to child health, and unless the physician of the future is able to cope intelligently with the "nerves" and "badness" of childhood he will suffer the humiliation of finding that his practice will be greatly limited, to say nothing of his sphere of usefulness. The day is past when we can divert worried parents with such phrases as "Wait till puberty and he'll outgrow it." "Take him out of school until his nerves are better"—"Don't cross him because he is a nervous child."

The behavioristic sciences of psychology and psychiatry are showing us beyond the peradventure of a doubt that the complaints for which such children are brought are symptomatic of a great variety of states of poor mental health, the nature and treatment of which cannot be got at too early. The physician in training and the physician in practice have for the most part ignored the contributions of

the behavioristic sciences because they have failed to recognize their usefulness in the practice of medicine. This has been partly due to the vague, involved and fantastic character of many of these contributions, and partly to the fact that medicine as a whole has confined its attention to a study of body quite apart from any consideration of the relationships of body to mind. Health can adequately be studied only from the standpoint of a thorough working survey of the individual as a whole. The principles underlying the behavior of child or adult are no more intricate and obscure than those underlying his biochemical activities. Both exist in such intimate relationship that it is impossible to evaluate any given health problem without careful consideration of facts based on both principles. In these articles I shall try to present a few principles that commonly underlie the "nerves" and "badness" of childhood.

The first step in becoming behavioristically intelligent concerning the "nerves" and "badness" of childhood is the realization of the fact that these conditions are symptomatic of strain in some sphere of the child's functioning. This strain may lie in discrepancy between individual capacities and environmental ambitions, or in faulty habit training, or in interparental disharmonies concerning policies of management, or in combinations of all these and other factors. The discovery of these strains takes a little time, and involves an orderly approach in the method of inquiry. It is so easy to jump at conclusions, blaming school for poor teaching, or parents for spoiling, without considering the intellectual stuff which the school is trying to teach, or the psychobiological stuff that parents are trying to train. We might outline the approach to an orderly inquiry concerning any given problem of poor mental health as follows:

I. Stuff out of which the individual is made:

Intellectual
Biological
Temperamental

II. Nature and effect of environment and training.

III. Factors precipitating unwholesome combinations of I and II.

In evaluating the ingredients of constitutional endowment, we have tests of science and tests of life; the latter are just as important as the former, although they cannot be checked by scientific controls. For example, in estimating the intellectual endowment of child and adolescent, psychology has given us the individual and group intelligence tests, and educational tests. In evaluating the biological endowment we have a wealth of nutritional and metabolic standards and tests of protein sensitization, etc. But science has given us no test for temperamental endowment, and yet from earliest childhood environment day by day crystallizes temperamental ingredients in a clear and un-

mistakable manner. Accordingly the problem of mental health is not constructively attacked by arguing whether heredity or environment is the determining fact in behavior. The real issue in every case is an individual matter dependent upon willingness to take time and patience to find out what kind of environment and training offers any given constitutional endowment of child and adolescent its best chance of growth and development.

Environments of home and school and other institutions of society are constructed on principles of what is good for average child material. Education until recent years developed on the principle of making children fit courses of study, instead of studying educational processes suitable to many different types of children. Until psychology gave us the Binet-Simon Intelligence test in 1911, teachers as well as doctors thought that the only intellectually limited child was the grossly feebleminded idiot, or imbecile, whose defective intelligence was objective enough to be diagnosed merely by inspection.

The intelligence test has been the subject of a great deal of criticism on the part of all sorts of people. This has been due partly to our inherent tendency to believe that each and every one of us is able to size up the intellectual ability of a human being from his own impressions of intelligence, and partly to the fact that we have never taken the time or trouble to look into the principles underlying the intelligence test. Again and again we hear many a teacher say, "I don't care what the intelligence test of this child is. I know that he could learn if he only wanted to." The result is that the slow, dull, and even defective child is pushed and prodded and tutored and sent to summer school, and promoted on trial with resultant strain followed by very serious consequences. For example, during the past year the Henry Phipps Psychiatric Clinic Dispensary, of the Johns Hopkins Hospital, had occasion to examine 79 junior high school misfits from two public schools. These children were referred for "easy tearfulness," "nervous stomach with nausea and vomiting during school hours," "headache and dizzy spells," "chronic truancy," "falling asleep in school," "petty stealing," "outbursts of tantrums," etc. In only ten of this number were found any physical handicaps associated with these complaints. Of the 41 boys in this group, only 4 were found to be of normal intelligence. The remaining 37 were trying to do junior high school work with an average intelligence of about ten years. Of the 38 girls examined, only 6 were of normal intelligence; the remaining 32 showed serious intellectual retardation. It was interesting to note also that coincident with increasing school strain there had developed in almost half of the entire group, such symptoms as bed-wetting, stammering, walking and talking in sleep, asthma, hives, eczema, fainting spells. The principles underlying these states

of poor health do not seem obscure or unreasonable. One would not put a Mack truck load on a Ford chassis; one would not insist on incasing a number six foot in a number four shoe; neither would medicine or education give a ten-year-old child an athletic program suitable for a fifteen-year-old. Yet departments of school health and departments of education apparently remain quite oblivious to the health problems that arise from intellectual misfitting. Such health problems are rarely ever referred to school doctors because teachers have found that the busy school doctor either has no time to seek the causes of such symptomatology, or else has no interest in it. Health education to the teacher means teeth, and tonsils, and eyesight, and hearing and nutrition, and posture. Symptoms of strain developing in the intellectual misfit are attributed as a rule by departments of education to natural perversity and laziness to be treated with disciplinary measures. The higher types of private schools protect themselves from the intellectual misfit by frequent and adequate use of the individual intelligence test, and educational tests.

Now it is perfectly true that the intelligence test like any other technic of scientific procedure can be used unintelligently. But one does not condemn the basal metabolic test or the x-ray because of the fallacy of their deductions in the hands of the unskilled and uninitiated. A wise and careful use of the Binet-Simon examination gives us an estimation of intellectual development that is as reasonably accurate as any other scientific evaluation of human functioning. One has only to follow the results of this psychological procedure in practice over a period of years to realize how well its results tally with the tests of life. It is conservatively estimated that 25 per cent of public school children are too mentally retarded to get beyond fourth or fifth grade work, and that another 10 per cent are wholly beyond their depth after sixth or seventh grade. The doctor who does not keep the possibility of such a condition ever in his mind in dealing with his small patients will find himself perplexed and baffled in many a case that comes to him. The history of the slow, dull, and even seriously retarded child does not show the gross evidences of delayed biological development that we are accustomed to find in the out and out defective. Dentition, walking, the beginning of words and sentence formation may be a little slow if one follows time relations carefully, but so great is the variation in these matters that a slight degree of slowness attracts neither the medical nor parental attention. If the parents are not particularly observant or of apprehensive temperament, the first intimation of intellectual retardation may be discovered only when the child begins school, and teachers complain that it is hard for him to catch on to their program. If classes are large, such a child not infrequently is passed on through the first few

grades without even attracting a teacher's attention. This is particularly true in schools where group intelligence tests, such as the Pintner-Cunningham are given to first and second graders.

Suspicion of the preschool child's limitations is very often dependent upon the temperament of a child. The quiet, docile, biddable, unaggressive little creature frequently has his backwardness charged up to timidity and shyness. Children who are annoyingly active and into everything and unresponsive to habit training are frequently diagnosed as "plain spoiled" by the physician. This is particularly true if the patient is an only child or the youngest. Many a mother is berated by the physician for spoiling her child without the slightest suspicion on the part of mother and physician that they deal with intellectually handicapped material which is unamenable to habit training quite satisfactory for a normal child. In such cases it is very helpful to spend a little time watching the reactions of this child in the office, in his playroom at home, and getting a parental report of the way the child behaves. In such situations one notices a marked defect in the attention span. One of the chief characteristics of normal intellectual growth is a progressive increase in the things which capture a child's attention and hold his interest. The retarded child flits or dashes from one object or activity to another, and makes a very poor showing in play, and even ordinary social contacts with his fellows. There is a distinct lack of purposefulness in his play—his use of toys—as well as a clumsiness of coordination in body movements. All these characteristics seriously handicap his training problem. There is another type of child in which suspicion of intellectual retardation must be considered, and that is the puny, whining, petulant boy or girl who bursts into empty tears and prolongs baby talk, hangs on to every slight illness with hypochondriacal complainings and seems definitely behind others of his own age in the process of growing up. Not infrequently one sees such a child standing out among his siblings. Here again parents are blamed for keeping this child a baby and otherwise making him what he is. The physician often forgets to ask himself why it is that these same parents have not spoiled and babied the rest of their flock. Experience in child health is coming to show more and more that behind the babyish, immature, hypochondriacally complaining child there is a constitutional endowment of definite intellectual or emotional instability which calls for special educational and training processes.

Here are early behavior reactions which may very easily pass unnoticed by the busy pediatrician. Their early recognition means everything to the child and his future. In coming upon such cases it is essential that such children be referred to a psychologist or psychiatrist with wisdom and common sense for a check-up not only on the child's

intellectual functioning, but also on the general reactions of his emotional life. If no psychological help is available, the physician can at least go slowly on advice and rigid prescriptions of training, feeling his way along. It is safer to err in giving such a child the benefit of doubt as to his constitutional capacities, and even if psychological and psychiatric assistance is available, there is many a time when the pediatrician must use his judgment as to whether this assistance is helpful or otherwise. For example, one can by no means always rely on one intelligence test or half a dozen. In many, many cases of intellectual retardation the mental age and the physical age of the child will run parallel sometimes up to 12 or 14 years of age before a discrepancy between the two becomes apparent with an intelligence test. No intelligence test should be interpreted without a thorough survey and evaluation of a child's general reactions to his entire environment. Too many a professional psychological tester bases his entire opinion of the intellectual status of a child or an adolescent on the intelligence quotient (the ratio between the mental age and chronological age). The tests of environment in the form of ability to cope with school work without strain, the degree of emotional maturity, evidenced in social contacts and the ability to hold one's own in group play and to show progressive sturdiness in other adaptations of environment must have equal weight in the evaluation of intellectual ability.

The treatment of the intellectual misfit is complicated first by the fact that his recognition is apt to be stubbornly resisted by parents, and second by the fact that school systems except in large cities have so little to offer the intellectually handicapped child. In large cities the special class, the opportunity class, and certain types of vocational schools called "pre-vocational schools" give these boys and girls a chance to find programs during school hours that they are able to grasp and enjoy. Unless a child is able to cope with school work his attendance at school is almost demoralizing. A continuous sense of failure is bad for all of us, and we react to it by trying to find satisfaction elsewhere. So does the intellectual misfit in school. Truancy, stealing, wandering away from home, setting fires, staying out late at night become much more attractive than sitting in a room where one is considered "dumb," and is teased and laughed at. In rural schools and the schools of small cities where there are no opportunities for handicapped children to have special educational processes, about all one can do is to try to see that teachers and parents fully realize that the child is doing the best he can, encourage him to get what he is able to grasp of ordinary grade work, and soft pedal his failures without constantly subjecting him to odious comparisons with his fellows. Such a child needs to feel that he has just as important a place in the

family circle and school community as if he could live up to all the standards set for his fellows. Withdrawal from school on special work permits as early as the law allows is far more beneficial to the mental health of such a child that compulsion to remain and try to graduate. Our compulsory education laws as they now exist are most unfair to intellectually handicapped adolescents. We should force no boy or girl to stay in school till 16 years of age if schools to which he has access afford no provision for an education that he can utilize. A simple job with the earning of a few dollars a week is of far more educational value in teaching such a youth good habit formation, the encouragement of real achievement, and a sense of responsibility, than any material he learns out of books. Moreover, insistence on the maintenance of formal educational standards is attended by the danger of breakdowns in stability that lay the foundations for nervous invalidism in later years ranging all the way from psychoneuroses to full-fledged mental disorders. Here is a field of preventive health which offers great opportunities for service to physicians in the realm of child hygiene. As doctors we have been too prone to relegate the behavior aspects of child health to the so-called educational disciplines. We need to have a reasonably intelligent understanding of what schools are doing and not doing. Teachers need our interest and constructive criticism in grasping and applying larger concepts of health education than they now possess. We are their logical source of helpfulness in dealing with the "nerves" and "badness" of childhood. If they find us indifferent and unintelligent toward the contributions of the behavioristic sciences of psychology and psychiatry, we cannot blame them for turning to other sources for advice. It is up to us to decide whether we shall let this large field of preventive medicine slip from our hands.

A DEFINITE SIGN PATHOGNOMONIC OF PARANASAL SINUSITIS

Preliminary Report

Maud Loeber, M.D., A. J. McComiskey, M.D., and
W. F. Henderson, M.D.
New Orleans, La.

IN REVIEWING the literature for more than the past fifty years, we were impressed with the fact that very little is noted in reference to puffiness about the eyes in relation to sinus infection. There is one very notable exception: de Schweinitz[1] in an excellent article on ocular signs of sinus diseases refers particularly to a "recurrent or fugitive edema and ecchymosis of the eyelids." In this article he describes a condition "which varies from a slight edema of the lid and flushing of the palpebral skin surface and surrounding area to a marked edematous swelling of red or brawny red color, usually associated with intense headache, and differs somewhat from the common edema of the lids which is so conspicuously present in acute ethmoiditis, antral and frontal sinus disease, in that it comes and goes—that is to say, presents itself in the form of attacks separated by intervals of time: it may be days, weeks, or even months of entire or comparative comfort; in other words, it is a typical recurring or fugitive edema and may be the only notable symptom of the affection. This is a common enough condition, although not always correctly interpreted and hence is briefly described." De Schweinitz quotes Robinson[2] who describes a "fugitive edema" of the eyelids; he says: "We often find those who are the victims of periodic headaches presenting a fugitive edema of the eyelids. We also find women during the menopause who have localized swellings of the hands and arms, swellings which are tender, pit on pressure, and are preceded by pain, but pass away after a few hours of exercise." De Schweinitz suspects that in a certain number of these cases unsuspected sinus disease was the factor, and suggests that all cases of periodic headache with fugitive edema of the eyelids associated with pain, with or without redness and with or without the appearances suggesting a subcutaneous ecchymosis, should include a thorough investigation of the sinuses. He also quotes Dunn[3] stating that recurrent edema of the upper eyelid was a symptom of nasal polypus.

In reporting the sign which we believe to be pathognomonic of paranasal sinusitis, it is not fugitive and need not be associated with sub-

From the Department of Pediatrics, School of Medicine, Tulane University; and the Touro Infirmary, New Orleans, La.

Submitted for publication, June 4, 1932.

Fig. 1-*A*.—R.M. Arrow indicates location of sign, quite noticeable on left side.

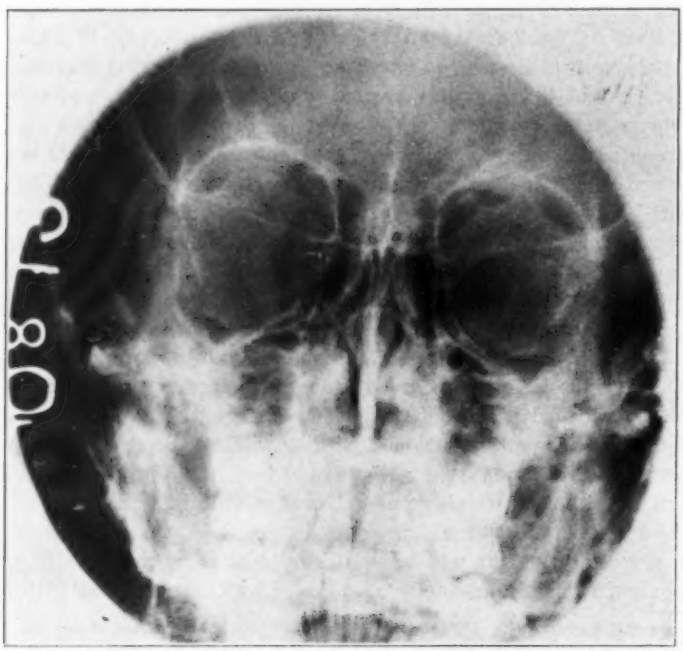

Fig. 1-*B*.—R.M. Clouding of both maxillary antra. Mucosa thickened and cavity of antra diminished. Remainder of nasal accessory sinuses normal.

Fig. 2-*A*.—L.S. Arrow indicates location of sign.

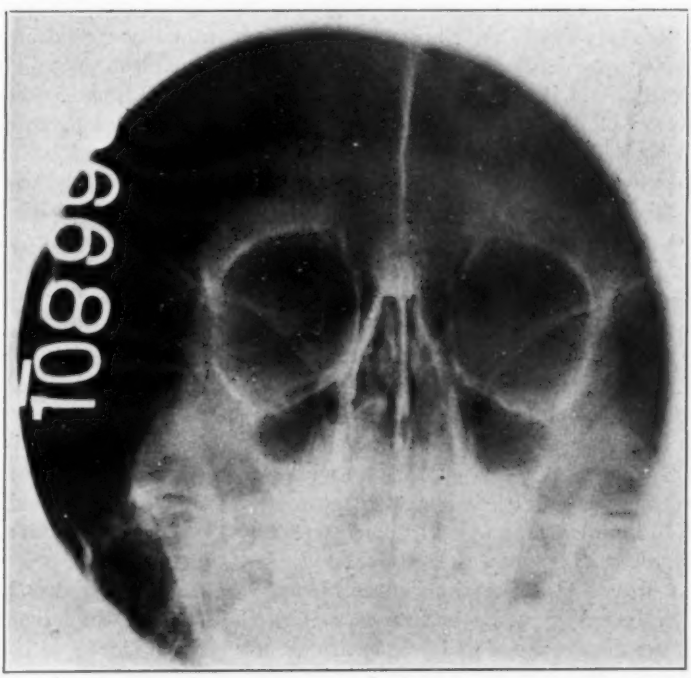

Fig. 2-*B*.—L.S. Moderate amount of clouding right maxillary antrum. Thickening of mucosa. Ethmoid cells on right cloudy, left normal. Frontal sinus undeveloped.

jective symptoms; in fact our attention originally was directed to the sign, despite the fact that the child could give us no leads by other signs or symptoms. We arrived at our discovery of its significant relation to sinus involvement more by elimination than by association with other manifestations.

As we continued our study, we found that the sign was also present in acute conditions as well as flare-ups in old conditions. The sign we wish to describe is present as long as any pathologic process is present in one or more of the paranasal sinuses, is more pronounced on that side which has the more disease, and is bilateral and symmetrical in conditions involving both sides of the face equally. It consists of a swelling in the loose subcutaneous tissue and redundant skin under the lower lid in its outer third. It resembles the appearance a face presents after a "good cry." It is not to be confused with ordinary edema of the lid but is rather a roughly, triangular area over the prominence of the malar bone. This sign can best be seen by turning the face so that the light strikes the face obliquely rather than directly. It cannot be clearly seen in strong direct light since the demonstration of the sign depends to some extent on the shadow cast by the swelling.

It has been our good fortune to be connected with a large out-patient clinic of a general hospital and to have many children attending it. To this clinic many social agencies engaged in the care and supervision of the underprivileged child bring their charges; in addition children are sent for examination from the institutions for dependents and delinquents, and it is primarily from these two large groups as well as the usual clinic population that this study was made; although we have corroborated our discovery in our private office work.

Jackson[4] and others have called attention to edema of the lids, though not this particular sign, and attempted to attribute it to renal disease, heart disease, malaria, syphilis, pyelitis, anemia, idiosyncrasy to some drug, such as arsenic, quinine, etc. We in turn have tried to explain the sign we are describing in like manner but met with disappointment. Recently noting that many of these children were malnourished and underweight and were suffering from a nasopharyngitis which recurred upon the slightest provocation, we suspected the paranasal sinuses. The authors conferred with one another, and each in his own field recorded his findings upon noting this clinical sinus sign independently one of the other. We have attempted to call attention to the frequency with which it is met in the routine examination of children, and to the high percentage of involvement of the ethmoids or antra, or both, where this puffiness is present.

Involvement of the sinuses, coincident with this edema, has been demonstrated by us on infants as well as children, for the ethmoidal cells are always found at birth[5] as are usually the maxillaries, although their appearance may be somewhat delayed until dentition.

TABLE I

AGE, YEARS	COLOR	SEX	HISTORY OF UPPER RESPIRATORY INFECTIONS					PARANASAL SINUSITIS SIGN				EAR, NOSE AND THROAT EXAM.			X-RAY EXAMINATION (SINUSES INVOLVED)									
			COLDS	TONSILLITIS	BRONCHITIS	OTITIS MEDIA	SINUSITIS	DEFINITE		INDEFINITE		CONFIRMS	NOT CONFIRM	NOT STATED	MAXILLARY		ETHMOIDS		FRONTAL		SPHENOIDS	
								RT.	LT.	RT.	LT.				RT.	LT.	RT.	LT.	RT.	LT.	RT.	LT.
9	W.	M.	+		+			+	+				v		+	+	+	+		+		
8	W.	F.	+	+	+	+	?	+	+					v	+	+	+	+			v	+	+
10½	W.	M.	+	+				+	+				v		+	+						
8	W.	M.	+	+	+			+	+			v			+	+	+	+				
1½	C.	M.	+	+	+			+	+					v	+	+	+	+			v		
9	W.	M.	−	−	−			+	+					v	+	+	+	+					
7	W.	M.	+					+	+			v			+	+	+	+			v		v
11	W.	M.	+	+	+		+	+	+			v			+	+	+	+					
5	W.	F.						++	+			v			++	+	+	+			v		v
11	W.	F.	+	+	+		+			+	+		v		+						v		
4	C.	M.	+	+	+			+	+				v		++	+					v		
9	W.	F.		+				++	+						++		++		++				
8	W.	M.	+	+	+		+	+	+			v			+	+	+	+		v			
5	W.	M.	+	+	+			+	+					v	+	+				v			
8	W.	M.								+	+			v	+	+	+	+					
6	W.	F.	+	+	+			+	+				v		+		+						
4	W.	M.						±	±				v		++	+	+	+	+	+		+	+
11	W.	M.	+	+			+			+	+	v			+	++				v			
9	W.	M.	+	+	+		+	+	+			v			+	++		+					
6	W.	F.		+				++	++			v			++	+	++		++	+		+	
4	W.	F.	+		+			+	+			v			+	+	+	+		v			
10	W.	F.		+				+	+			v			+		++	+					
8	W.	M.		+				+	+			v			+	+	+	+					
2½	W.	M.	+	+	+	+		+	+				v								v		
9	W.	M.		+	+	+		+	+						++	+	+	+	+				

Table I: Explanation of signs used in table.
+indicates presence of pathologic condition and sign.
++indicates marked presence of pathologic condition and sign.
−indicates negative pathologic condition, but presence of sign.
v indicates result of consultation with ear, nose and throat department, or x-ray department.

We have found this sign to be a most valuable aid in arousing our suspicion of sinus involvement, since it occurs early in the disease before the development of other distressing sequelae such as bronchitis and asthma. These sequelae develop in a young child prone to aspirate secretions dripping from the nasopharynx. It has been our observation that the puffiness is greater in the mornings probably due to poorer lymphatic drainage of that area due to position as has been demonstrated by noting the route of elimination of lipiodol following injection of an antrum, as recently pointed out by Pfahler[6]. We have frequently demonstrated the sign in old, long-standing chronic cases; it is also found in those cases exhibiting only a diseased lining membrane without free pus. As the patient improves, whether through surgical drainage of the sinuses in severe cases, or removal of the tonsils and adenoids with conservative treatment of the infected sinuses, the above sign becomes less noticeable until it completely disappears.

Table I gives the results of twenty-five cases as they came through the clinic routinely, the sign being present. It should be stated that in no instance did the child aid us with usual clinical symptoms of sinus involvement, such as pain on bending over, headache or dizziness.

In the majority of the cases the swellings were bilateral, sometimes one slightly more marked than its fellow. In these we have found sinus involvement on each side, with a more marked swelling on the side where the involvement was greater or more extensive. In one case (an adult, however) the sign was demonstrated unilaterally, and the findings proved to be a marked unilateral involvement of one maxillary antrum on the same side as the sign.

SUMMARY

A roughly triangular swelling of the portion of loose skin beneath the outer third of the lower lid over the malar prominence is pathognomonic of pathologic process in one or more of the paranasal sinuses of that side.

This has been confirmed by a rhinologic examination as well as demonstrated by roentgenography.

The affected sinuses need not be filled with pus to produce the sign, but it is found as well in those sinuses exhibiting only a diseased lining membrane.

Amelioration or disappearance of the sign was coincident and proportional with the local improvement of the sinuses involved.

CONCLUSION

A sign has been described which we consider as pathognomonic of paranasal sinusitis. We believe that the recognition of this sign permits of an early diagnosis of sinusitis which may offset the sequelae of a chronic focus of infection.

The authors are indebted to Dr. Harry L. Bacal, resident Fellow in Pediatrics at Touro Infirmary, for the tabulation of our findings.

REFERENCES

1. De Schweinitz, G. E.: Tran. Am. Ophthalmol. Soc., 46th annual meeting, page 477, 1910.
2. Robinson: Brit. M. J. May 12, 1888, as quoted in Ref. 1.
3. Dunn, John: Am. J. Ophthalmol. **9**: 134, 1892.
4. Jackson, Edward: A Manual of the Diagnosis and Treatment of Diseases of the Eye, Philadelphia, 1907, p. 459, W. B. Saunders Co.
5. Coffin, Lewis A.: Sinusitis in Children; the Nose, Throat, and Ear; Jackson and Coates, 1929, W. B. Saunders Co., 126-139.
6. Pfahler, George E.: A Demonstration of the Lymphatic Drainage From the Maxillary Sinuses, Am. J. Roentgenol. **82**: 352, 1932.

1430 Tulane Avenue.

GROWTH PROBLEMS AND THEIR RELATION TO ZONES OF NORMAL PHYSIOLOGIC REACTIONS

WILLIAM PALMER LUCAS, M.D., HELEN BRENTON PRYOR, M.D., CRAWFORD BOST, M.D., SAXTON T. POPE, M.D., AND MARION C. HENDERSON, A.B.

SAN FRANCISCO, CALIF.

THE physical approach to growth problems of children has brought us to search for new standards of growth and for new measures of development. Generalizations fail us when we are face to face with the problem presented by the individual child. How far can the body assist in the application of scientific precision to the problem of growth? Can we measure the relationship of a child's indefinite and temporary complaints to the child as a whole by these new critical methods of approach? These ever pressing questions have dominated our study of constitutional type from the first. To translate the child from the "general," the "normal," the "average" to the particular, to his own individual variations, reactions, et cetera, and still leave him related to his past and to his future, has been the challenge of this study to us.

We have already demonstrated the value of body measurements from which can be calculated "ideal" weights for individual children. By "ideal" weight we mean merely to differentiate from average weight, the proper weight for the individual child taking his body build into consideration. The Baldwin Wood Tables give average weights for height and age. These average weights are labeled "normal" by school nurses and others who report to parents that their children are under or over normal weight. We have found that at least 30 per cent of our children vary significantly from this average because of their individual constitutions or body builds. A child with a large bony framework should weigh more than the average for his height and age, whereas a child of the same age and height with a slender framework must of necessity have an individual normal weight or ideal weight for build which is much less than his lateral type playmates. Dr. Franzen[1] emphasized the importance of skeletal dimensions in determining variations from average weight.

WEIGHT AND BODY BUILD

Mathematical analyses have shown that body weight is more closely related to width of shoulders, chest and hips than it is to height. Children inherit slender or stocky or intermediate types of body build from their parents and are heavier or lighter than average in exact proportion as they are broader or slenderer than average for their age. We

Submitted for publication, June 27, 1932.

have worked out a width-length index which measures the relative breadth or body build of a child. Many elaborate methods of classifying body build have been worked out, but the very simple way of merely comparing width to length of body appears to be adequate. The widest diameter of the crest of the pelvis divided by the height yields an index of build.

This width-length index increases directly with the relative breadth of the child, since it expresses the simple relationship of each body to itself. Empirically, it can be said that a low index means a slender child with small bones, and a high index means a broad child with large bones. Calculating the width-length index measures the amount of variation from the height-weight-age table averages that should be allowed for body build before diagnosing overweight or underweight. If a child's width-length index is small, he should weigh less than the height-weight table indicates, and if his index is large, he should weigh more than the table indicates.

Obstetric calipers or anthropometric spreading or sliding calipers may be used to measure diameters. No other special equipment is necessary and the time required to calculate the index is very little.

Each child has an "ideal" weight which can be calculated for his build. For example, an eight-year-old boy is 52½ inches tall and weighs 57¼ pounds. His "normal" weight according to the height-weight-age table is 63 pounds, so he is labeled 9 per cent underweight by the school nurse. However, he is a slender-built boy with narrow diameters, and the ratio of the width of his body to his height shows he is 5½ per cent narrower than the average eight-year-old boy in his social group. Ideal weight calculated for his particular bony framework is found to be 56 pounds. He is then found to be 1¼ pounds above his ideal weight.

A six-year-old boy is 44 inches tall and weighs 48 pounds. His "normal" weight on the height-weight-age table is 44 pounds so he is labeled 9½ per cent overweight by the school nurse. But this boy is broad built with large diameters, and the ratio of the width of his body to his height indicates that he is 7 per cent broader than the average six-year-old boy in his social group. His ideal weight for his body build is found to be 49 pounds, so he is actually 1 pound under his ideal weight.

Table I gives average indices on 3,400 cases by age and sex.

Standard deviations indicate that weight varies from the height-weight-table average twice as much as the width-length index varies from the average at each age when both are measured in per cent. For example, if a boy, aged seven years, has an index 151 (4.5 per cent less than the average seven-year-old index, 158) his weight could be ($2 \times 4\frac{1}{2}$) 9 per cent less than the height-weight table average and still be normal for his frame. If he is 48 inches tall his normal weight, according to the height-weight table, is 53 pounds, but since his hips

are so slender he could weigh 9 per cent less, or 48.3 pounds and be normal for his frame. Or if a girl, aged four years, has an index of 176 (8 per cent larger than the average four-year-old index, 163) her weight could be 16 per cent greater than the height-weight table average and still be normal for her frame. If she is 40 inches tall her normal weight, according to the height-weight table, is 36 pounds, but since her hips are 8 per cent wider than the average for her age she could weigh 16 per cent more than 36, or 41.7 pounds, and be normal for her frame.

TABLE I

AGE	MALE	FEMALE
0-1	173	175
1	168	172
2	167	171
3	166	166
4	162	163
5	161	161
6	159	159
7	158	159
8	158	159
9	158	159
10	158	160
11	157	161
12	157	162
13	156	163
14	155	163
15	155	164
16	154	164

We[2] published these figures last year feeling that this method helps to make the measurement of nutrition a concrete individual matter.*

The advantage of working out the ideal weight for the particular child has been the basis of extending our studies into other phases of growth and the reason for searching out the particular variations found in different constitutional types.

Draper's[3] ideas of constitutional types and the division of these types into various panels—anatomic, physiologic, immunologic, and psychologic—have challenged us; and, although we do not agree fully with the ultimate conclusion of his thesis "that the constitution of an individual determines in large part the type of disease which will become his particular hazard," we do feel that an individual's particular constitution is responsible for certain trends and that this is more likely than Draper's conclusion. Again we find that the zonal conception of constitutional trends more nearly represents the problems of growth than the fixed or static idea of Draper's. Growth never follows a straight line as we saw in the older plotting of weight curves.

*In using the width-length index we have found two places where the percentage variation from index for age should not be doubled in calculating percentage deviation for weight. (1) At both extremes on the index scale (the very slender indices 130-140 and the very broad indices 185-195), and (2) in cases where the child is too tall or too short to appear on height-weight-age scale. In these extreme cases the *same* percentage deviation from the index should be allowed for weight deviations instead of double the percentage. For example, the rare ten-year-old boy whose width-length index is 130 (17 per cent narrower than the average 158) should weigh 17 per cent less than the height-weight table average weight but probably not 34 per cent less. This is based on clinical judgment from experience of the last three years.

Individuals who stand just inside the edges of the normal growth zone may, to all appearances, vary but slightly from those found just outside the normal zone. Those outside the present accepted normal zone have an ideal normal for their own build and constitution which fits their type as well as the older standards fit those within the so-called normal zone.

However, one often finds children who are outside the normal zone as well as those within the normal zone who show, without any specific ailment, variations in growth activities which create definite problems. Positive relationships among physical, physiologic and mental traits may be easy to point out in the majority of cases, but we must not fail to consider the negative relationships which demand definite analysis and which reveal a mass of tangled interrelationships not shown up in any other way.

COMMON INDEFINITE SYMPTOMS

Every pediatrician is familiar with the child who comes with the particular complaint of increased fatigue, i.e., more tired from less activity than the normal child shows and the fatigue lasting. There are all types of fatigue but the type we are discussing is the one that results from a given set of circumstances which ordinarily in the normal child would cause little or no fatigue and that lasts longer than in the normal child.

Another symptom common to these children is an increased susceptibility to infections. Here again, their infections occur more frequently, last longer and are usually more severe than one finds in the child with normal reactions when exposed to the same infections.

Other obscure growth problems, such as failure to gain in weight and strength, stubborn adiposity, loss or lack of appetite, digestive upsets such as vomiting, diarrhea, or constipation, an increased irritability, increased instability, often a change in character and temperament, i.e., smiles and tears seem nearer the surface, temper tantrums and sulkiness flash out more unexpectedly and with less provocation, may be grouped into the familiar symptom complex which these children so often present. There is nothing specific the matter with these children. The physical examination does not bring out sufficient cause to explain the symptoms and yet we see these children often, and many times fail to meet their need. These frequent failures determined us to attempt at least to use other methods of approach to their problems.

Applying mathematics to such clinical data is a form of statistical technic which may act as a new kind of microscope in revealing hidden clinical truths. The placing of numerical values upon the relationships between as unlike traits as the physical, physiologic and mental has helped us to develop the proper technic of correlation. It is possible to assign numerical values to certain types of clinical material by reducing all data to terms of percentage variation from average for each age, sex,

group at hand. Then the correlation[4] coefficient can be computed. Correlation is expressed as a coefficient and is significant in terms of its probable error. For example, the relationship between the number of red cells in the blood and the standing height could be found by determining the average number of red cells of the group of people under consideration and their average standing height. Each individual would be rated in terms of percentage variations from these averages for his age, sex, group, and the application of the Pearson correlation formula would measure the amount of the relationship for the group studied. It is not enough to present averages because we find averages merely point out trends. We find that statistical analysis shows not only the existence of a relationship but also its extent or amount which involves an exact measure of variability and the amount of overlapping. We have found considerable light thrown on growth problems by being able in a measure to map out some of the zones of normal physiologic activity, or, in other words, to allow with greater degree of accuracy for the marginal elasticity in growth zones and to capture more definitely the degree of agreement between traits studied.

We have not attempted to develop anything new but simply to use those physiologic tests which experience has generally accepted as well established. One of these is the basal metabolic rate.

METABOLIC STUDIES

Our metabolism studies were begun three years ago. The Benedict Roth apparatus has been used throughout. Ages of the children have ranged from three to eighteen years. Calculation of rate has been referred to the Benedict Talbot scale where it has been applicable. Where it has not (i.e., above 38 kg. for boys and 58 kg. for girls), the Harris Benedict and Aub-du Bois standards have been used as supplementary. Early in the work other standards were suggested and employed, but abandoned because of their greater complexity and more restricted usefulness.

Reliability of standards for children and the possibility of testing younger children in a really basal state have both been questioned. Practically the test has offered a few difficulties but not so many as had been anticipated. The average child of intelligent parents seems recently to have achieved a real indifference—objectivity at least—toward the methods of medical investigation, and, for most of them the bogeyman has departed from the oxygen tank. Difficulty is to be anticipated principally from the wilful child, and the younger child who has been through an inhalation anesthesia. But even these, if permitted to watch another patient taking a metabolic test, will usually lose their fear; or if not, the fear will have worn itself out on a subsequent morning.

Reliability of standards is one of the more abstruse problems of metabolic study which has never been perfectly answered. We feel that we have shed some light on this problem.

Two determinations that checked were done on each child before the test was accepted. Rechecking of basal metabolic rates was done at three-month intervals on very high and very low metabolisms.

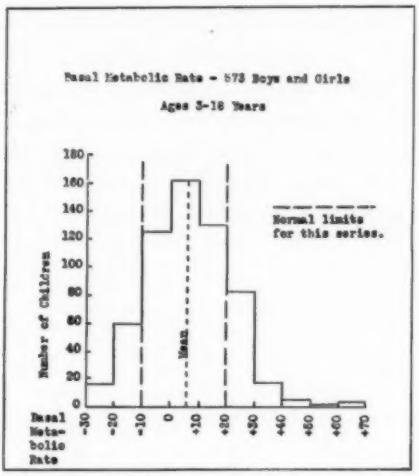
Basal Metabolic Rate - 573 Boys and Girls
Ages 3-18 Years
Number of Children
Normal limits for this series.
Mean
Basal Metabolic Rate
-30 -20 -10 0 +10 +20 +30 +40 +50 +60 +70

Fig. 1.—Frequency distribution of first acceptable basal metabolic rate for 573 children. There is a symmetrical distribution of cases around a mean of plus 6.4 per cent which pushes the upper normal limit to plus 20 per cent for this series.

TABLE II

TABLE OF DISTRIBUTION OF BASAL METABOLIC RATE SHOWS NO SEX DIFFERENCE OF SIGNIFICANCE

BASAL METABOLIC RATE DISTRIBUTION BY SEX
573 CHILDREN, AGES 3 TO 18 YEARS

Sex	Basal Metabolic Rate										Total
	-30	-20	-10	0	+10	+20	+30	+40	+50	+60	
Male	11	30	65	62	66	26	7	1	0	2	270
Female	6	30	60	100	64	26	11	3	2	1	303
Total	17	60	125	162	130	52	18	4	2	3	573

TABLE III

TABLE OF DISTRIBUTION OF FIRST BASAL METABOLIC RATE SHOWS MARKED AGE DIFFERENCE

FIRST BASAL METABOLIC RATE BY AGE GROUPS AND SEX

Age Group	Basal Metabolic Rate—Males										Totals
	-30-	-20-	-10-	0-	+10-	+20-	+30-	+40-	+50-	+60-	
3- 5			6	3	10	5				2	26
6- 9	4	10	22	26	29	15	7	1			114
10-17	7	20	37	33	27	6					130
Totals	11	30	65	62	66	26	7	1		2	270
Age Group	Basal Metabolic Rate—Females										Totals
	-30-	-20-	-10-	0-	+10-	+20-	+30-	+40-	+50-	+60-	
3- 5		2	4	8	12	4			1		31
6- 9	1	7	27	45	33	13	8	3		1	138
10-18	5	21	29	47	19	9	3		1		134
Totals	6	30	60	100	64	26	11	3	2	1	303

During these three years 576 patients have been tested, and 1,027 metabolic rates calculated. There have been two proved thyroid hyperplasias, which were operated upon, and one known cretin. These three were not included in the present figures. The remainder, however, have been occult deviations, where deviations existed. Figures for the first accepted rates on the first 573 patients are as follows: If the heretofore acceptable limits of normal rates be considered as plus and minus 10 per cent, there have been 287 normal, 371 with positive deviations and 77 with negative deviations. The graphic distribution of our total series of first basal metabolic rates (573 cases) is seen in

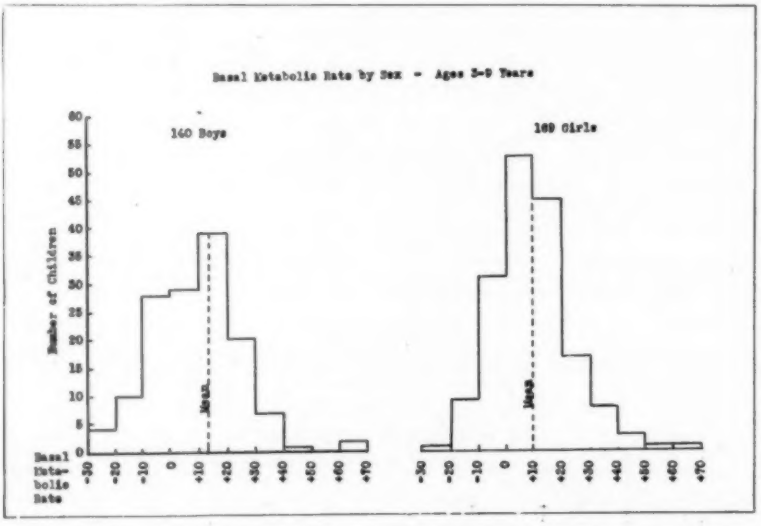

Fig. 2.—Frequency distribution by sex for preadolescent age group shows the mean basal metabolic rate for 309 children is above the usually accepted upper limit of normal. The normal zone for this series is zero to plus 22 per cent.

Fig. 1. It is evident that plus 10 per cent cannot be considered the upper limit of normal since the mean for the whole series is plus 6.4 per cent and since the graph shows a normal frequency curve. It is therefore necessary to stipulate plus 20 per cent as the upper normal limit. The distribution then becomes 425 normals and 79 with positive deviations against 77 with negative. Then 74 per cent of our series are within the new normal limits, approximately 13 per cent deviate above normal and 13 per cent deviate below normal. (See Table II.)

For analysis of results by age and sex groups on first basal metabolic rates (see Table III). The mean basal metabolic rates on our series are shown in Table IV.

TABLE IV

26	boys	ages	3	to	5	years	plus 18.0 %
31	girls	ages	3	to	5	years	plus 10.5 %
114	boys	ages	6	to	9	years	plus 8.7 %
138	girls	ages	6	to	9	years	plus 9.14%
130	boys	ages	10	to	17	years	plus 0.46%
134	girls	ages	10	to	17	years	plus 2.48%

We have grouped the ages, preadolescent three to nine years and adolescent ten to seventeen years. At the preadolescent age 140 boys and 169 girls show the same range of from minus 30 to plus 50 basal metabolic rate (see Fig. 2). The mean is plus 13 per cent for boys and plus 10 per cent for girls at this age with a fairly normal distribu-

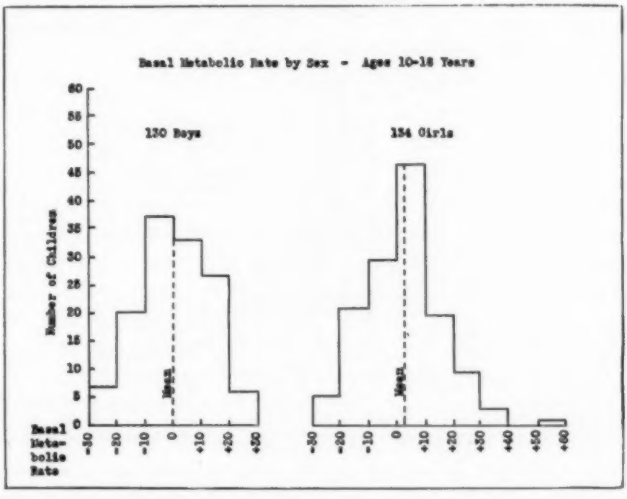

Fig. 3.—Frequency distribution by sex for adolescent age group shows the mean basal metabolic rate to be for boys zero and for girls plus 2.5 per cent. The normal zone is the usually accepted minus 10 per cent to plus 10 per cent.

tion of both sexes. At the adolescent age level 130 boys and 134 girls show normal distribution of basal metabolic rates. (See Fig. 3.) The boys range from minus 30 per cent to plus 30 per cent and the girls range from minus 30 per cent to plus 40 per cent. The mean for boys is 0 and for girls it is plus 2.5 per cent.

There is a negligible sex difference in the basal metabolic rate but a very marked age difference. (See Tables V and VI.) Analysis of our data year by year shows a marked difference to occur with adolescence. Preadolescent children have higher basal rates than adolescent children. The mean basal metabolic rate for our series of 309 cases both sexes aged three to nine years is plus 11.58 per cent and for 264 cases both sexes aged ten to seventeen years is plus 1.43 per cent.

For ages ten to eighteen a deviation of minus 10 per cent to plus 10 per cent on the Benedict Talbot scale represents the normal zone for our series of 264 children. This corresponds to the usually accepted standard.

For ages three to nine years, because the mean basal rates are plus 10 per cent for girls and plus 13 per cent for boys, the normal zone deviations become 0 and plus 20 per cent for the two sexes together on the Benedict Talbot scale. (See Fig. 4.)

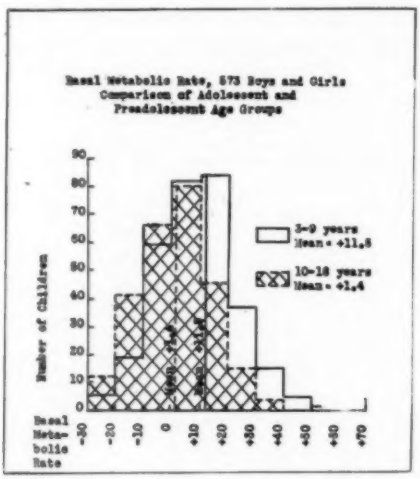

Fig. 4.—Graphic comparison of distribution of basal metabolic rate for adolescent versus preadolescent children. A correction factor of minus 11.5 per cent is suggested for preadolescent age group basal metabolic rates referred to the Benedict Talbot scale.

Instead of suggesting a new standard for the preadolescent age group we offer the use of a correction factor in interpreting basal metabolic rates referred to the Benedict Talbot scale. For ages three to nine years the basal metabolic rate as calculated minus 11.5 per cent would have the same significance in indicating therapy as the uncorrected rate on the older children.

TABLE V

TABLE OF DISTRIBUTION OF BASAL METABOLIC RATE FOR BOYS SHOWS MUCH HIGHER RATES FOR PREADOLESCENT THAN FOR ADOLESCENT AGE GROUP

BASAL METABOLIC RATE BY AGE GROUPS

270 BOYS

Age Group in Years	Basal Metabolic Rate										Total
	-30	-20	-10	0	+10	+20	+30	+40	+50	+60	
3- 9	4	10	28	29	39	20	7	1	0	2	140
10-18	7	20	37	33	27	6	0	0	0	0	130
Total	11	30	65	62	66	26	7	1	0	2	270

In Topper's[13] data on 38 children a serial study of basal metabolic rate is presented for ages ten to sixteen years. Her figures show a definite increase in basal metabolic rate before and during early puberty but a fall after puberty is established. The rise occurs at ages twelve to thirteen in girls and at ages thirteen to fourteen in boys, which is consistent with the fact that girls attain puberty earlier than boys. The statement is made that the average basal metabolic rate at age eleven is higher in girls than in boys probably because puberty has already begun in girls at this time.

TABLE VI

TABLE OF DISTRIBUTION OF BASAL METABOLIC RATE FOR GIRLS SHOWS MUCH HIGHER RATES FOR PREADOLESCENT THAN FOR ADOLESCENT AGE GROUP

BASAL METABOLIC RATE BY AGE GROUPS

303 GIRLS

Age Group in Years	Basal Metabolic Rate										Total
	-30	-20	-10	0	+10	+20	+30	+40	+50	+60	
3- 9	1	9	31	53	45	17	8	3	1	1	169
10-18	5	21	29	47	19	9	3	0	1	0	134
Total	6	30	60	100	64	26	11	3	2	1	303

In view of the fact that in our own series the mean basal metabolic rates are consistently higher the younger the children, we question the prepubescent rise. The point is made by Topper that the pulse rate rose with the rise in metabolism and fell coincidently with the return of the basal metabolic rate to normal. The fact that both the normal pulse rate and rate of respiration are progressively higher the younger the child seems confirmation of our high basal metabolic rates in young children.

The correlation between basal metabolic rate and body build index when both are reduced to percentage variation from the mean for the age sex group is -0.237 ± 0.026. This is a negative correlation and is well validated statistically. It means that slender-built children have higher basal metabolic rates than broad-built children. That as children's build grows slenderer their basal metabolic rates get higher and as children's build grows broader their basal metabolic rates get lower. Also, that degrees of activity of the thyroid gland are related directly to degrees of linearity of body build.

Our feeling is that these children with basal metabolic rate deviations are not true cases of endocrine disturbance but that during certain growth periods their basal metabolic rate is disturbed. This disturbance may be due to repeated infections or other physiologic or psychologic strains which create definite disturbances in their autonomic nervous systems as well as in their endocrine systems. This is especially true during the preadolescent period. We have found that the basal metabolic rate is disturbed in quite a large proportion of the children

who manifest the symptom-complex already given. The disturbed metabolism is undoubtedly closely connected with their symptoms since a correction of their basal metabolic rates very definitely restores the children to their normal growth processes.

Many preadolescent children with complaints of fatigue, susceptibility to infection, et cetera, and all the symptoms that are usually found grouped around those two, show basal metabolic rates well outside the usually accepted normal zone described. We had arrived at the conclusion clinically, that plus 10 per cent is probably within the normal zone and that plus 20 per cent is nearer the actual limit of the normal range of increased metabolism. When abnormal metabolic rates have been brought back to within the normal zone of metabolic function, the symptom-complex is easily corrected, whereas without the appreciation of the metabolic disturbance and its correction, these symptoms tend to continue over a much longer period. We do not claim that this deranged metabolism is permanent, but that it is simply an indication of disturbed physiologic function, temporary in character, or perhaps is merely one of those growth phenomena brought about by too rapid or too slow growth. Its correction simply shortens the time during which this symptom-complex remains a disturbing element. Nature itself would probably correct, in time, not only the metabolic rate but also the other symptoms that grouped together in these children. They rarely die from this group of symptoms but their lives are distinctly under par, and anything but a searchingly fundamental attempt to get at the cause of the trouble fails. Most of the obvious things such as change of environment, rest, diet, hygiene, etc., have all been tried without any lasting results. These various measures result in only temporary relief from the symptoms which return at the slightest provocation. A corrected metabolism has brought about, in most instances, a permanent abeyance of these symptoms. When a return of symptoms occurs we find that the metabolic rate has again gone beyond the normal zone. The cases to be given will show these metabolic disturbances in relation to the symptom-complex.

BONE AGE X-RAYS

Whenever a constitutional inadequacy was suspected we have had joint x-rays made to determine bone age. We have followed the method described by Shelton[5] last year and compared our plates with his standards allowing a deviation of six months either way before diagnosing retardation or acceleration of ossification. In a small highly selected series of 51 cases, 28 boys and 23 girls, ages thirteen months to fifteen years, we found 13 that deviated more than the six months allowed. Nine of them were boys and four were girls which is in line with Dr. Todd's[6] statement that throughout childhood and adolescence a larger proportion of boys than of girls shows defects of a temporary

nature in growth and development velocities. Only two of our thirteen were definite endocrinopathies, one a cretin and one a eunuchoid giant.

Dr. Todd has pointed out that the rate and velocity of epiphyseal union as recorded in x-ray studies provide a reliable register of progress. He says: "On a single examination they give a definition of status. Serial or periodic roentgenographic examination of the skeleton provides a measure of progress. This physical expression is not an academic finding but an exceedingly important quantitative indicator of constitutional health or defect and may be applied with confidence in the assessment of developmental growth." We feel that repeating physical measurements or exercise tolerance tests at intervals also provides measures of progress.

EVALUATION OF EXERCISE TOLERANCE

One of the most difficult evaluations to make is that of the degree of fatigue existent and how much stress to lay upon this in our treatment. After trying out several tests we have chosen the Schneider exercise tolerance test.[7] This test was devised by Schneider to estimate fitness for flight. It has a numerical evaluation for a series of factors which is supposed to give an estimate of one's physical fitness. It is probably primarily meant to test cardiac response but actually gives us an estimate of general muscle, nervous system, cardiac and, to a lesser extent, respiratory fitness.

Briefly, the exercise tolerance employed in the office is one which determines six factors and assigns values to them. The factors are these:

1. The cardiac rate, while resting in the supine position.
2. The cardiac rate, two minutes after the assumption of an easy standing position.
3. The difference between these two rates.
4. The acceleration of rate over the standing occasioned by stepping five times in fifteen seconds from floor to knee height.
5. Time of return to preexercise standing rate.
6. Difference in systolic blood pressures, supine and standing.

Scoring has been done as suggested by Dr. Schneider, by giving a mark of 3 to each of the six factors or grading on a scale up to 18, from 8 to 18 being within the normal zone. One cannot achieve more than the maximum 18 points, the so-called perfect score. Aviators were permitted to fly with scores as low as 8. But in the negative side, poor scores range from 8 to –3. We have never obtained a score below minus 3. Poor scores give us an estimate of the degree of cardiac weakness not necessarily connected with cardiac disease, but perhaps the result of general muscle and nervous states. We have made very slight revisions in using the test and only as dictated by the necessity to reconcile the circumstances of adulthood and childhood. The reproduced comparison test may be too close, for one cannot always predict

safely that responses for adult and child will be alike both in degree and kind. Roughly, however, it seems to be true. One test simply gives the present condition; repeated tests give us the progress either toward a normal rating or toward an increasing cardiac and muscle failure or nervous instability.

The Schneider test is performed as follows:

The patient rests supine for five minutes, or until two successive 20-second pulse rates are identical. Brachial systolic blood pressure is recorded. Patient then assumes an easy standing position. After two minutes the pulse rate and brachial systolic blood pressure are recorded. Next, the patient places one foot on a step, 18 inches in height, and, at given signals, steps up and down with the other foot. The signals are arranged so that he raises his body through the distance (18 inches) five times in 15 seconds. The examiner places his fingers on the radial pulse immediately at the conclusion of the exercise period, and counting for 15 seconds, estimates the rate per minute. Subsidence to the preexercise standing level is awaited, and the time noted. If at the end of the 2 minutes the rate is still elevated above it, the pulse is counted for a quarter of a minute, and it is recorded. The results may now be computed, scoring as follows:

A. Reclining pulse rate.		*B. Pulse rate increase on standing.* 0-10	11-18	19-26	27-34	35-42
50- 60	3 points	3	3	2	1	0
61- 70	3	3	2	1	0	–1
71- 80	2	3	2	0	–1	–2
81- 90	1	2	1	–1	–2	–3
91-100	0	1	0	–2	–3	–3
101-110	–1	0	–1	–3	–3	–3

C. Standing pulse rate.		*D. Pulse rate increase immediately after exercise.* 0-10	11-20	21-30	31-40	41-50
60- 70	3 points	3	3	2	1	0
71- 80	3	3	2	1	0	0
81- 90	2	3	2	1	0	–1
91-100	1	2	1	0	–1	–2
101-110	1	1	0	–1	–2	–3
111-120	0	1	–1	–2	–3	–3
121-130	0	0	–2	–3	–3	–3
131-140	–1	0	–3	–3	–3	–3

E. Return of pulse rate to standing normal after exercise.

Seconds	Points	
0-30	3	
31-60	2	
61-90	1	After 120, 2-10 beats above normal = –1
91-120	0	After 120, 11-30 beats above normal = –2

F. Systolic blood pressure rise on standing.

Change in m.m.	
Rise of 8 or more	3 points
Rise of 2-7	2
No rise	1
Fall of 2-5	0
Fall of 6 or more	–1

In attempting to apply the test to children, it at once became apparent that the varying pulse rates and blood pressures of childhood would require consideration. Average rates as presented in Abt's System *V. IV, pp. 220 and 225,* were examined and it was found that three groupings could be made, without serious distortion. They are as follows: One of four to six years on the basis of a rate of 90. A second of six to ten on the basis of 80. A third of ten to fourteen on the basis of 70. Since 70 is the average adult rate, for the third group, the adult score is used. The revisions for Groups 1 and 2 are simply nonfractional projections of the original score. They are as follows:

Revision No. 1

Age 4 to 6

		B. Pulse rate increase on standing.				
A. Reclining pulse rate.		0-13	14-23	24-33	34-44	45-54
65-77	3 points	3	3	2	1	0
78-90	3	3	2	1	0	–1
91-103	2	3	2	0	–1	–2
104-117	1	2	1	–1	–2	–3
118-131	0	1	0	–2	–3	–3
132-145	–1	0	–1	–3	–3	–3

		D. Pulse rate increase immediately after exercise.				
C. Standing pulse.		0-13	14-26	27-39	40-52	53-65
78-90	3 points	3	3	2	1	0
91-103	3	3	2	1	0	0
104-116	2	3	2	1	0	–1
117-129	1	2	1	0	–1	–2
130-142	1	1	0	–1	–2	–3
143-155	0	1	–1	–2	–3	–3
156-168	0	0	–2	–3	–3	–3
169-181	–1	0	–3	–3	–3	–3

E. Return of pulse rate to standing normal after exercise.

Seconds	Points	
0-30	3	
31-60	2	
61-90	1	After 120, 2-10 beats above normal = –1
91-120	0	After 120, 11-30 beats above normal = –2

F. Systolic pressure standing compared with reclining.

Change in m.m.	Points
Rise of 6 or more	3
Rise of 1-7	2
No rise	1
Fall of 1-4	0
Fall of 5 or more	–1

Revision No. 2

Age 6-10

A. Reclining pulse rate.		*B. Pulse rate increase on standing.*				
		0-11	12-21	22-30	31-39	40-48
59-69	3 points	3	3	2	1	0
70-80	3	3	2	1	0	–1
81-91	2	3	2	0	–1	–2
92-102	1	2	1	–1	–2	–3
103-113	0	1	0	–2	–3	–3
114-124	–1	0	–1	–3	–3	–3

C. Standing pulse rate.		*D. Pulse rate increase immediately after exercise.*				
		0-11	12-22	23-33	34-44	45-55
70-80	3 points	3	3	2	1	0
81-91	3	3	2	1	0	0
92-102	2	3	2	1	0	–1
103-113	1	2	1	0	–1	–2
114-124	1	1	0	–1	–2	–3
125-135	0	1	–1	–2	–3	–3
136-146	0	0	–2	–3	–3	–3
147-157	–1	0	–3	–3	–3	–3

(For E. & F. refer to Revision No. 1)

It was also apparent that the uniform height of step could not be used, since it exacted a disproportionate effort of the shorter patients. Eighteen inches (the specified adult requirement) was found to be just short of average knee height from the ground. It was decided, therefore, to take some fairly stable measurement on the children and vary the height of the step for each on that basis. The fibular length—fibular head to external malleolus—was selected and an adjustable stand contrived.

Scoring for the "E" section is unchanged from the original.

To date (April 20) the exercise tolerance of 214 patients has been computed, with 92 repeats. The series will not be subjected to complete analysis until its total is considerably more representative.

Scores have been examined, however, on the first 189 cases, and, even if no exact conclusions can be drawn, the results are suggestive. The group studied was that which has previously been defined, and a few routine cases of suspected and proved endocarditis and valvular heart disease. The mean test score for the group as a whole was 10.2. For males 11.1, and for females 9.41. Perfect scores were made by two patients, a male and a female. The lowest score was minus 2. Scatter diagrams showed no particular trend.

It has been noticed in our series that more patients whose body build was of the linear type stood above the mean than might be expected by sheer chance alone.

When on reviews the exercise tolerance of individual patients it is found to mirror one's clinical impressions with surprising accuracy, and has the distinct advantage of confirming one's suspicions and assigning to them a particular figure or number which has been found a great help in clarifying the situation for parents. Results have been

seen in their purest form, perhaps, in frank instances of valvular disease, and hypofunctional glandular states. One of the perfect scores was made by a boy with the signs of aortic regurgitation, entirely compensated. In another boy with aortic regurgitation and failing circulation we watched, pari passu, a dwindling score and heart muscle reserve. Less definitely cardiac and probably much less dependable are the results in instances of autonomic instability of the cardiovascular sort, but even these variable and flighty figures themselves are helpful. Occasionally they unmask a thyroid hyperactivity—frequently they suggest the needed form of therapy. On the whole this test has proved of considerable value in estimating the amount of activity a child should be allowed or the amount of rest we must impose. It removes the personal equation from the estimation of an individual's progress and substitutes a numerical evaluation which should be more exact.

ANOREXIA AND BODY BUILD

We[8] have noted a general similarity among children whose main complaint is anorexia. Body measurements on 184 of these children showed width-length indices grouping decidedly in the slender-built zone. Eighty-two per cent of them were well below the mean index for their age sex groups. Obstinate constipation, poor nutrition, fatigue, irritability and high basal metabolic rates were characteristic of these 184 children. Analysis of the usual diets of these children showed that many of them were being given a high proportion of fat in their daily rations with the hope of fattening them.

We have recognized the necessity of feeding children according to their powers of digestion and their individual anatomic needs. Determination of body type by measurement gives a good indication of these two things. We have had satisfactory clinical results with special diets worked out for divergent anatomic types. In general the slender-built do best on low fat, high vitamin diets.

HEREDITY AND PERSONALITY STUDIES

The hereditary and psychologic aspects of these cases are ones that cannot be neglected. One must get as much of the family history as possible. This is rather difficult to obtain from the average American family, whose memories cover only from one to two generations. We have worked out a simple questionnaire which attempts to get information on the body build and prevailing psychologic and immunologic tendencies of the child's forebears back as far as the grandparents, at least. The parents were asked to estimate their child's disposition in terms of placid, medium or highstrung, his school standing, his activities and his general family reactions. Therefore, in so far as personality traits can be measured by subjective ratings, we have some comparisons of personality and body build in normal children.

TABLE VII

TABLE OF DISTRIBUTION FOR SUBJECTIVE RATINGS OF CHILDREN'S DISPOSITIONS. TWICE AS MANY CHILDREN ARE CONSIDERED HIGHSTRUNG AS ARE CONSIDERED PLACID BY THEIR FAMILIES. THE PRESCHOOL CHILDREN ARE EVENLY DIVIDED PLACID AND HIGHSTRUNG

DISTRIBUTION BY SEX AND AGE GROUPS

Age Group in Years	Placid			Highstrung		
	Male	Female	Total	Male	Female	Total
0-5	9	9	18	10	11	21
6-9	7	11	18	30	45	75
10-18	16	15	31	14	28	42
Total	32	35	67	54	84	138
Per cent	48.0	52.0	100.0	39.0	61.0	100.0

Questionnaires have been analyzed for 205 children, ages three months to fifteen years.

138 children were labeled highstrung, and
67 children were labeled placid by parents.
(For distribution see Table VII.)

There were 119 girls and 86 boys.

84 or 70 per cent of the girls were labeled highstrung.
35 or 30 per cent of the girls were labeled placid.
54 or 63 per cent of the boys were labeled highstrung.
32 or 37 per cent of the boys were labeled placid.

Some interesting age differences are to be noted. Under five years of age approximately an equal number of children were placid and highstrung, and there was no sex difference in distribution of traits. (See Fig. 5.) At ages six to nine years (beginning of school days) four times as many children were considered highstrung as were considered placid (75 to 18). The ratio of 4 to 1 holds for both sexes.

45 girls were highstrung.
11 girls were placid.
30 boys were highstrung.
7 boys were placid.

For the adolescent group 42 children were considered highstrung and 31 children were considered placid. A larger proportion of adolescent girls than boys was considered highstrung.

28 girls were highstrung.
15 girls were placid.
14 boys were highstrung.
16 boys were placid.

We know subjective ratings by parents are of limited value and the method is not one of precision in measuring a trait but the spectacular increase in nerve tension reported at a certain age level commands attention. Because of the great increase in the proportionate number of children considered highstrung in the age group six to nine, one must consider the possible effect of the first formal school training.

In view of the work that has been done, we have made no attempt to find relationships between physical traits and intelligence. We are interested in studying relationships between physical and personality traits. Anthropometric standards have been established by international agreement and are objective. The definition and measurement of personality traits are much more difficult, but recent progress has been made.

The psychologists[9] who measure personality traits have abandoned subjective estimates or ratings and have perfected many scales and tests to be used as measuring instruments. Much research has been devoted to the determination of validity and reliability. Use of stand-

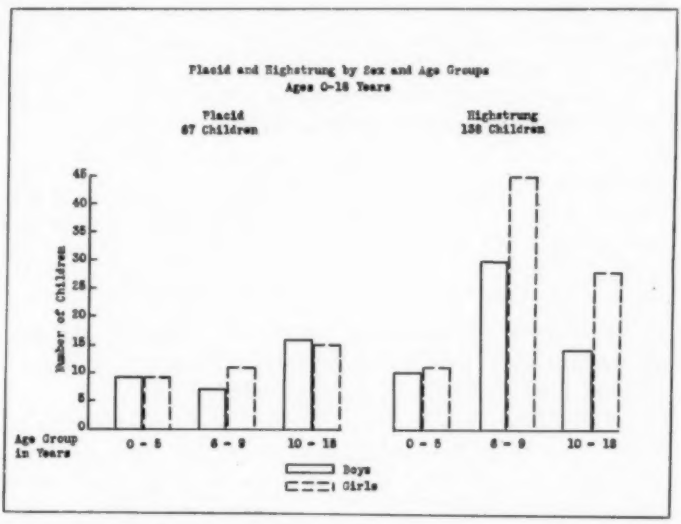

Fig. 5.—Graphic distribution of children's dispositions shows more than four times as many preadolescent children are highstrung as are placid. More adolescent girls than boys are highstrung.

ardized validated tests may be considered a method of precise measurement for personality data and test scores can be correlated with physical measurements on a basis of percentage variation from average for the group.

We have used the Almack Sense of Humor Test[10] to measure sense of humor. It consists of 13 cartoons, 34 jokes and a list of 50 characters to be rated in terms of very funny, fairly funny and not funny. Numerical scores on this test have been compared with body build, in our adolescent age group and in general our broad-built children have higher scores than our narrow-built of the same age sex group. Older children have higher scores than younger. Extraverts have higher scores than introverts.

In general the lateral type tends to be the extravert and the linear type the introvert. The number of highstrung children increases markedly with the beginning of school age. These studies have emphasized for us the importance of estimating the psychologic factors that enter into the constitutional make-up of the child, an aspect pediatricians cannot afford to neglect while studying the physical aspects of a child.

To quote again from Dr. Todd's article on adolescence[6]: "Looking into the future, I believe that this method of physical approach will prove to be applicable to problems of the mind as well as to those of the body. I can do no more than allude to the probable great significance of further studies on the developing cerebrum of the child. At the moment the problem can be roughly sketched. Localization of function within the cerebrum is established beyond a doubt, but the mind acts as a unit. This whole-mindedness is the mental counterpart of physical coordination in bodily responses. The body reacts as a unit in terms of its training, the mind in terms of its experience.

"The pediatrician can never hope to succeed in his study of adolescence except by feeling his way steadily from the physical aspect on which he has some secure basis of understanding to the less explored regions of the mind and of the spirit."

CEPHALIC INDEX AND BODY BUILD

What significant relations exist between the mental and physical traits? Does a high brow actually denote intelligence or is the long headed man farsighted and of superior intelligence?

Literature[12] is full of assertions that long-headedness implies mental superiority and broad-headedness implies mental dullness. Cephalic index has been used by many to assess the intellectual quality of whole groups of people. Writers in anthroposociology have pointed out that long-headedness is characteristic of superior races in general and of outstanding leaders within a given race and that Aryan and Nordic superiority was a result of high proportions of long-headed people. A gradual increase in size of the head can be demonstrated to parallel cultural evolution over thousands of years but within a contemporary group head size does not increase with intelligence. Race, sex and body type influence both size and shape of head more than intelligence does.

We have compared cephalic indices with width-length indices for 545 children, 277 girls and 265 boys, and have found long-headedness roughly parallel to slender-body build and broad-headedness roughly parallel to stocky-body build. That is, in general, the cephalic index increases as the width-length index increases. When we compared degrees of long-headedness with linearity of body build the Pearson Coefficient Correlation is for boys $r = +0.213 \pm 0.039$; for girls $r = +0.185 \pm 0.039$. These are both significant correlations. The rela-

tionship between build and shape of head is closer in boys than in girls for this series.

Glands of internal secretion presumably play an important part in determining both physical and personality traits if we can argue backward from results of endocrine imbalance. For example, the cretin after thyroid therapy is a changed creature in both physical appearance and temperament. Interrelations proved to exist in extreme cases of deviation from average raise the question of the recognition of these relations in normal children.

Case histories to be presented in Part II of this paper will demonstrate more clearly than any descriptive account of methods used what has seemed to us the possibilities of these studies—suggestions of procedure in treatment that become of dynamic importance to the child. We have met, of course, the perfectly justifiable criticism of this research—Why all this fuss and labor and expense in a doctor's office practice to establish what most of us can reach, upon the basis of our clinical experience alone?

All we can say to such criticism is that our experience has satisfied us that these more elaborate and exact methods have given us a point of view and an approach to these individual problems of children that have clarified our understanding of the individual and have helped us to more exact lines of treatment. No one without clinical experience could, of course, either appreciate fully the problems of these children or could they apply these more exact methods. However, we know that often clinical experience alone leads to the successful handling of these cases. But the clinician who successfully handles these problems by intuition cannot share his method of approach with his contemporaries. We only claim that in our studies we have gone further in more adequately meeting the needs of these problems in children than when we depended entirely upon our clinical experience.

CONCLUSIONS

1. That the physical approach to growth problems necessitates the definition of new normal zones to replace some of the fixed standards of the past. The modern objective has changed from an attempt to make all children conform, to an attempt to enable each child to develop according to his own individual capacities.

2. Application of simple statistical technic to clinical data is a new kind of microscope which reveals heretofore hidden clinical truths.

3. Simple body measurements, such as width of shoulders and hips combined with height and weight, yield a satisfactory evaluation of a child's build or constitutional type.

4. Studies of basal metabolic rate in children show a much higher normal rate for the preadolescent age group than the former standard allowed.

5. The establishment of new normal zones for basal metabolic rate gives a better idea of the internal glandular systems and perhaps of the autonomic nervous systems in problem children.

6. X-rays taken for bone age are of material assistance in determining skeletal status and growth progress.

7. Exercise tolerance tests assist occasionally in clarifying a diagnosis when fatigue is a prominent symptom or when there are cardiac difficulties, and have definitely aided in outlining more exact treatment and in estimating the improvement or lack of improvement obtained by the treatment.

8. Certain relationships between hereditary and psychologic traits and constitutional type or body build are suggested by a mathematical study.

REFERENCES

1. Franzen, Raymond: Physical Measurements of Growth and Nutrition, Am. Child Health Ass'n, 1931.
2. Lucas, W. P., and Pryor, Helen B.: J. A. M. A. **97**: 1127, 1931.
3. Draper, George: Human Constitution, Philadelphia, W. B. Saunders Company, 1924.
Idem: Disease and the Man, New York, Macmillan, 1930.
4. Kelley, Truman L.: Statistical Method, New York, Macmillan, 1923.
5. Shelton, E. Kost: J. A. M. A. **96**: 759, 1931.
6. Todd, T. Wingate: Am. J. Dis. Child. **43**: 533, 1932.
7. Schneider, E. C.: J. A. M. A. **74**: 1507, 1920.
8. Lucas, W. P., and Pryor, Helen B.: Am. J. Dis. Child. **41**: 249, 1931.
9. Loofbourow, G. C.: The Validation of Test Materials for the Discovery of Problem Behavior Tendencies in Junior High School Boys. (Not published.)
10. Almack, M. D., and Almack, J. C.: A Sense of Humor Test, Stanford University Press, 1928.
11. Bernreuter, Robt. G.: The Personality Inventory, Stanford University Press, 1931.
12. Paterson, D. G.: Physique and Intellect, New York, Century Company, 1930.
13. Topper, Anne, and Mulier, Hannah: Basal Metabolism of Normal Children: The Puberty Reaction, Am. J. Dis. Child. **43**: 327, 1932.

490 Post Street.

LOBAR PNEUMONIA IN INFANTS AND YOUNG CHILDREN

CLIFFORD G. GRULEE, M.D., AND PHILIP MULHERIN, M.D.
CHICAGO, ILL.

THE subject of lobar pneumonia in infants and young children is one which has received very little attention in the last few years. There is a general opinion in the medical profession that lobar pneumonia at this age is rather infrequent and that bronchopneumonia is the ruling lung condition. In a small service over a period of ten years we had the interesting experience of having more cases of lobar pneumonia than of bronchopneumonia. We, therefore, felt that it might be of value to look into the clinical picture of lobar pneumonia as shown in the hospital cases to see if the popular ideas regarding that condition were tenable.

One should not draw too far-flung conclusions on any group of cases because in the nature of things any group is in a way selective. In order to get the proper perspective of this group we can say that all the cases of lobar pneumonia treated by the Pediatric Staff at the Presbyterian Hospital, Chicago, over a period of ten years from January 1, 1922, to December 31, 1931, are included. It should be said too with respect to age that the facilities for the care of children are about equally divided between those for children under eighteen months and those for children over eighteen months. The patients come largely from the Central Free Dispensary in connection with the Hospital and from Infant Welfare Stations. Occasionally patients are admitted from orphan asylums, and there is the usual proportion of private cases that one would expect to meet with in a private hospital. The latter group constitutes about 20 per cent of the total.

Hospitalization of patients makes possible a more accurate diagnosis and gives better control of the patient from a therapeutic standpoint. On the other hand, it is altogether likely that many patients would come to the hospital only if they had reached a certain degree of severity. The probabilities are that the cases in this report are of a more severe nature than would be met with in private practice in the home.

ETIOLOGIC CONSIDERATIONS

After going carefully over the histories including autopsy findings, we found that in the ten years in question, we had a total of 116 cases of lobar pneumonia. Sixty-nine, or approximately 60 per cent, were

From the Children's Department of the Presbyterian Hospital, Chicago.
Submitted for publication, July 5, 1932.

in boys and 47, or approximately 40 per cent, were in girls. Of these 116 cases, 29, or 25 per cent, occurred in the first year, 66, or 57 per cent, between the first and fifth years, and 21, or 18 per cent, between the sixth and twelfth years. The number each year varied from six to nineteen. The seasonal variation was as follows: From June first to August thirty-first 19 cases, or 16.4 per cent; from September first to November thirtieth 22 cases, or 18.9 per cent; from December first to February twenty-eighth 37 cases, or 31.9 per cent, and from March first to May thirty-first 38 cases, or 32.8 per cent. It is noted that the incidence during the winter and spring quarters was higher than that for the summer and fall. In fact, nearly two-thirds of the cases occurred from December first to May thirty-first. In Chicago, this represents decidedly the cold portion of the year. It is rarely warm in May and never continues so. The springs are cold and wet and usually associated with cold winds off the lake. The temperature may be very variable during this period. No attempt was made at any bacteriologic diagnosis from these cases since it is altogether likely that contamination would have been so great that no conclusions could have been drawn.

ONSET

The history of onset is of course derived from information obtained from the mother or attendant. In 50 cases this was said to be sudden, while in 64 it was insidious. In 2 cases it was not definitely stated. One of these two had had a cough and fever for two and one-half weeks; the mother had then been informed that the child had pneumonia. The other was an infant two weeks old that began vomiting and was admitted for that condition only. Of the 50 that began suddenly, 47 are known to have been associated with fever usually described as high; while in three a mention of fever was not made. One came in as an appendicitis case because of abdominal pain and vomiting; in 2, coughing was the only symptom noticed.

Temperature elevation was noted at onset in 106 cases. There were 10 in which no reference was made to temperature at the beginning. Five of these have already been mentioned in the preceding discussion on the variety of onset. Of the other five, one began with rapid respirations, one with vomiting and cough, one with diarrhea, one with cough, vomiting and dyspnea, and one was admitted complaining of loss of appetite and vomiting.

Cough was present at onset in 77, and in 39 there was no cough mentioned. The cases without cough were distributed almost equally in three age groups, 12 coming in less than one year, 15 in one to five years, and 12 in five to twelve years. Vomiting was present in 39 of the patients at onset; of these, 25 were associated with cough, while 14 were not. It is interesting to note in this respect that vomiting

was the initial symptom in all age groups. In the age group of less than one year there were 6 cases, from one to five years 24 cases, from six to twelve years 9 cases. Contrary to expectation this would indicate that vomiting was a symptom to be expected rather in the older than in the younger children, since the percentage of cases starting with vomiting increased with age.

Convulsions were noted at onset in only 4 of the patients. There was one case in which the mother described convulsions which were interpreted by the attending physician as "fright." Of the 4 patients, 2 were less than one year old and the other 2 three and one-half and seven years, respectively. In only one of these were convulsions repeated, and they were associated with symptoms of meningeal irritation, but lumbar puncture failed to reveal anything pathologic except a high sugar content, 95 mg. per 100 c.c.

SYMPTOMS

Of all the symptoms of lobar pneumonia the one probably of which we hear most is the *cough*. In children especially it is this symptom toward which the most therapeutic measures are directed. It is, therefore, interesting that in this group of 116 cases of lobar pneumonia there were 7 in which there was no history of cough and no notation of cough at any time during the course of the disease. There were 18, including these 7, in which there was no cough at any time after admission to the hospital. In 56 others the cough was mild, in 33 severe, and in 9 distressing. One must remember that these estimates are purely subjective and are made by attendants who are not related to the child; in other words, the nurses and physicians in the hospital. They may, therefore, be regarded as a fairly accurate estimate of the cough. We see from this that probably only 9 really demanded therapeutic measures for control of this symptom.

Diarrhea.—Diarrhea was present in 17 cases but in only 2 was it at all severe. Of the 15 mild cases that cleared up immediately after admission all seemed to be due to the effect of castor oil given before entrance. In 2 cases where diarrhea was severe, there was a history of castor oil having been given for some days before admission. We see, therefore, that diarrhea in this series was practically nonexistent as a symptom of lobar pneumonia and in all probabilty was due to unnecessary medication.

Urinary Findings.—Albumin was present in 39 cases and casts in 15. In only 2 of the cases in which albumin was found was this large in amount. One of these was a case which was complicated by hemorrhagic nephritis and the other by encephalitis. Of the 15 cases showing casts, in only 3 were more than an occasional one noted. Two of these 3 had large amounts of albumin; the other was without complication and never showed any albumin.

White Blood Count.—Table I shows to best advantage the white blood count.

TABLE I

AGE	UNDER 10,000	10,000-15,000	15,000-20,000	OVER 20,000	HIGHEST
0-1	0	7	7	15	54,000 – 3 mo.
1-5	2	12	13	39	61,000 – 3 yr.
6-12	0	1	8	12	39,000 – 11 yr.
Totals	2	20	28	66	

The lowest count was in an infant fourteen months old, the count being 7300; the highest was in a child three years old—61,000. Over half the cases showed a white count of over twenty thousand and over three-fourths of the cases showed a white count of over fifteen thousand.

Fever.—The course of the fever is not so characteristic as is found in the adult. There were 51 (43.9 per cent) cases which ended by crisis; of these 8 patients were under one year of age, 31 from one to five years, and 12 from six to twelve years. There were likewise 51 which ended by lysis, of which 12 were less than one year of age, 31 from one to five years, and 8 from five to twelve years. Of the remaining cases 9 had a pseudocrisis, of these 6 came down later with a true crisis, and 3 ended by lysis. There were 16 patients with intermittent fever, 10 cases of which ended by crisis and 4 by lysis; 1 remained high at the time of release and 1 died. There were 6 cases where fever was low, that is, under 101° F. Of these patients, 1 died, 1 was released before the course terminated (incidentally this child was in a very serious condition), and 1 other developed a delayed resolution. We see, therefore, that the end by crisis is not as usual as in the adult; that the end by lysis is much more frequent than at the later age, and that intermittent fever and low fever occur in a fair proportion of the cases. An analysis of the cases ending by crisis as compared with those ending by lysis according to age brings out a surprising agreement. There is a slight tendency for the crisis to be a little more frequent as the age advances.

Other Symptoms.—A few other symptoms may be noted. Chills were found in 9 cases, 6 of which were in children between one and five years and 3 between six and twelve years.

Abdominal pain was likewise noted in 9 cases during the course of the disease; of these 6 were between one and five years and 3 between six and twelve. It is rather interesting to note that there was a decided difference in the symptom of abdominal pain according to the various years. There were no cases where abdominal pain was noted during the years 1922, 1923, 1924, 1928, 1929, and 1930. In 1925 there were 2 cases; in 1926, 3; in 1927, 1; and in 1931, 3. It is

possible that this may have something to do with the character of the prevalent respiratory infections of that year. On the other hand, it may be due to pleurisy which in certain years is doubtless more frequent than in others. We have noted no definite connection between the physical findings and the presence of abdominal pain in this series of cases.

PHYSICAL FINDINGS

Lobe Involvement.—Lobe involvement is best shown in Table II.

TABLE II

LOBES	UNDER 1 YR.	1-5 YR.	5-12 YR.	TOTAL
Rt. Upper				
No. of cases	14	12	5	31
Per cent	45.1	38.7	16.1	
Alone				24
Associated				7
Rt. Middle				
No. of cases	7	7	0	14
Per cent	50.0	50.0	0	
Alone				8
Associated				6
Rt. Lower				
No. of cases	7	16	8	31
Per cent	22.5	51.3	25.8	
Alone				24
Associated				7
Lt. Upper				
No. of cases	6	8	0	14
Per cent	42.7	57.1	0	
Alone				7
Associated				7
Lt. Lower				
No of cases	8	26	8	42
Per cent	19.0	10.0	62.0	
Alone				31
Associated				11

In addition to these cases of lobe involvement there were 4 cases of central pneumonia. An accurate diagnosis was made in only 2 of these 4 cases, in one of which the symptoms had existed for fourteen days and the crisis occurred three days after the diagnosis was made by x-ray. In another case the clinical diagnosis of pneumonia was made after the x-ray revealed a right lower lobe involvement. In two others the findings were negative, but pneumonia was suspected and in each case the x-ray substantiated the suspicion. In one of these the crisis occurred three days after the x-ray was taken and in the other the day after. In addition to these cases, there was one case which was uncertain. The clinical course was that of a lobar pneumonia which ended by lysis between the ninth and thirteenth day with resulting recovery. The x-ray findings were unsatisfactory and it is

possible that this case should be classified as a bronchopneumonia rather than as a lobar pneumonia. In all there were 17 cases, or 14.6 per cent, in which more than one lobe was involved.

Statistically it is impossible to say just which of the findings were present first. At the time the diagnosis was made, dullness was always present and in most instances there was at least a small patch of bronchial breathing. Crepitant râles were almost never noted except in older children. Mucous clicks were occasionally heard. The presence of dullness with bronchial breathing was in nearly all instances the indication for a roentgenogram. In 85 of the 116 cases, the roentgenogram confirmed the clinical diagnosis. We do not consider that the absence of the confirmative roentgenogram with positive clinical findings is sufficient cause to exclude the diagnosis. In the first place one may take an x-ray at a time when the density is not sufficient to show; again there may be a thickened pleura; or the pneumonic process may be situated behind the heart, making it difficult to picture on the film. Sometimes the physical findings are present several days before the x-ray confirms our suspicions. We, therefore, cannot regard the x-ray in lobar pneumonia in children as an absolute diagnostic measure, but we do think it is of the greatest help.

Complications.—Otitis media was associated with lobar pneumonia in this series in 32 cases. In 13 it preceded the onset of the pneumonia and in 19 it occurred during the course. Of these children, 8 were less than one year, 23 were between one and five years, and only 1 between six and twelve. The distribution of otitis media is interesting as is shown in Table III. We see that the incidence of

TABLE III

YEAR	NO. OF CASES	PER CENT	YEAR	NO. OF CASES	PER CENT
1922	1	16.6	1927	2	13.3
1923	1	10.0	1928	3	50.0
1924	3	16.6	1929	8	42.1
1925	1	12.5	1930	3	25.0
1926	4	40.0	1931	6	50.0

otitis media is decidedly greater in some years than in others. This again suggests that the type of respiratory infection which is prevalent during that period is responsible for the presence or absence of a complicating otitis media.

Empyema was a complication in only 3 of the 116 cases. We do not know why this figure should be so low. Unresolved or delayed resolution was present in 3 cases, a pyelocystitis in 3, and hemorrhagic nephritis developed in 2.

The other complications noted were subcutaneous abscesses, 1 case, meningitis, 1 case, and encephalitis, 1 case.

Prognosis.—In these 116 cases there were 7 deaths. In the group of 29 patients under one year of age 6 died, a death rate of 20.8 per cent; of the 66 children from one to five years of age, 1 died, a death rate of 1.5 per cent; of the 21 children between six and twelve years none died.

The *first* death was in a child four months of age who was brought into the hospital because of vomiting. While in the hospital he developed a lobar pneumonia and died on the eighteenth day after the onset of vomiting, sixteen days after admission. The autopsy showed a lobar pneumonia in the lower lobes with fibrinous pleurisy, congenital pyloric stenosis, and anemia. The *second* death was in an emaciated male infant of eight months in whom cough and weakness were the serious features, both of them interfering with his feeding. He died on the nineteenth day of his illness, five days after admission. No autopsy was obtained. Case 3 was a seven-weeks'-old baby who died on the twenty-fifth day of illness, four days after admission. It developed signs of meningitis on the third day, but lumbar puncture was unsuccessful because of a bloody tap. No autopsy was obtained. Case 4 was a fifteen-months'-old baby who died sixteen days after admission. While the autopsy showed this case to have an extensive bronchopneumonia in the right lower lobe with an abscess of this lobe, it seems altogether likely that the cause of the illness had been a lobar pneumonia with abscess formation in the right lower lobe and secondary bronchopneumonia. Case 5 was an eight-months'-old baby who died on the eighth day of his illness, seven days after admission. There were no special findings in this case and autopsy was refused. Case 6 was a six-months'-old baby who had had a diarrhea for three weeks and was cyanotic when brought to the hospital. The child died within twenty-four hours. Autopsy was refused. Case 7 was a seven-months'-old male infant who evidently had a migrating lobar pneumonia and died five days after admission on the eighth day of the disease. No autopsy was obtained.

The analysis of these cases brings out the fact that there was no death in a child over fifteen months of age. Many of the other children were in extremely serious condition when brought to the hospital. One of the deaths was undoubtedly due to the presence of a lung abscess, another was probably due to meningitis, and a third was evidently affected by the presence of a congenital pyloric stenosis which could not be operated upon because of the child's general condition. The fourth was in a child that had had a severe diarrhea for three weeks and died in less than twenty-four hours after admission.

TREATMENT

In the treatment of these cases no specific therapeutic measures of any sort were employed either in the form of serum or drug. The children were given nursing care, emphasis being laid on fresh air, but

not cold air. Food was given them at regular intervals to the amount which they would take, but it was never forced except on special order of the attending physician. The only thing that was given that might be termed as a general medication was a small quantity of alkali which was given in only 16 cases. Therefore, treatment was nearly entirely symptomatic. Cough medicine was given sometime during the course of the disease in 16 cases. The drug most used for coughs was atropine. This was used in 11 of the 16 cases and was the only drug used for the cough in these cases. It was given very often in some cases and only occasionally in others. Of these, one patient died. Elixir terpine hydrate was given in 3 cases. In 2 of these it was given but once and no other medication was given. In the other case, a twelve-year-old child, elixir terpine hydrate was given four times and codeine seven times. In one other case one dose of codeine was given and in another case five doses of elixir alurate.

It is seen from these figures that in only one case, that of the twelve-year-old child, was there any adequate medication for cough. Atropine was used rather extensively, but other forms of drug therapy for the control of cough may be said to have been very rarely used.

As a rule nothing was done to control the temperature, but aceto-salicylic acid was given in 14 cases for this purpose. In 28 cases some cardiac stimulation was used. This was usually caffeine or camphor oil, occasionally digitalis. In these 28 cases a stimulant was given but once in 5 cases. Of the other 23 the number of doses varied from two to over seventy-five. In this group it is interesting to note that 7 patients died. Thus this group contains all the fatal cases.

We would like to mention here that oxygen was occasionally used but only as an emergency measure, and we had no experience with the use of the oxygen tent.

The insistence in the treatment of lobar pneumonia in our wards is that the child shall be left alone; that it shall be carefully nursed and that food be given at regular intervals; that it shall have plenty of fresh air, but that so far as possible all drug treatment shall be dispensed with. We believe that the failure to give drugs in this series of cases accounts for the lack of gastrointestinal complications which we think it will be admitted, were exceedingly few.

TEMPERATURE AND PRECIPITATION IN RELATION TO THE MORBIDITY RATES OF POLIOMYELITIS

JOHN A. TOOMEY, M.D., AND MYRON H. AUGUST, M.D.
CLEVELAND, OHIO

IN A RECENT analysis[1] of the seasonal changes occurring in poliomyelitis epidemic years, we were impressed by the fact that in this locality, low precipitation and a relatively high temperature were present in the spring months of April, May, and June in all but one of the epidemic years.

In the first Swedish epidemic, Bull[2] stated that the whole summer was unusually warm, but made no mention of precipitation. Frost[3] studied the following epidemics: Mason City and Grundy Center, Iowa, in 1910 where 86 cases occurred; Cincinnati, Ohio, in 1911 with 106; Covington, Kentucky, with 44; Buffalo, New York, in 1921 with 281, and nearby Batavia with 23 cases. In Mason City, there was a period of high temperature during March and the first half of April with a variable curve for the latter half of the month. There was a low precipitation rate for April, May, June, and July. In Grundy Center, there was a decrease in precipitation from February to August with a definite March temperature increase. Frost dismissed the connection between temperature and precipitation since more cases occurred in August, the month of maximum rainfall. In fact, there were more cases in the wetter second half of the month than in the drier first half.

In Cincinnati and immediate surroundings, the temperature in 1911 was above in January, February, May, and June, normal in March and slightly below in April while the precipitation was above normal in April and below in the other growing months of May and June.

Most authors feel that temperature and precipitation can be ignored since the months when the case morbidity was highest were often the wettest months of the year and because the height of the temperature and the apex of the epidemic curve do not correspond. The weather conditions during an epidemic could be ignored if the factors causing the disease arose during the months of highest disease incidence.

It has been thought by some that poliomyelitis is a disease of gastrointestinal origin which might possibly follow the ingestion of foodstuffs. If the causative factors were associated with or coincidental with growth, then the weather of the early spring or growing months both as regards temperature and precipitation might have some bearing on the ripening

From the Department of Pediatrics, Western Reserve University, and the Division of Contagious Diseases of the City Hospital.
Submitted for publication, July 11, 1932.

of fruit, etc., in such a manner as to prepare the stage for an epidemic which could later occur irrespective of the precipitation at the time of the epidemic. The question was interesting enough to investigate and the weather statistics and case morbidity rates of poliomyelitis in 15 cities were studied.

The precipitation rates of the growing months of April, May, June, and July were added together for an arithmetical normal. The same was done to the temperature readings. In this addition, the decimal points were dropped to make comparisons easy in the tables. For example, 459 of precipitation is equivalent to 4.59 inches of rain. In the tables, an average is placed under each city both for temperature and for precipitation. In Los Angeles, the growing season is obviously earlier by at least a month so that the months considered here were March, April, May, and June (Tables I, II, and III).

The meteorologic data from the 15 cities referred to are contained in the following paragraphs:

Buffalo, New York. Nineteen ten, 1912, 1915, 1924, 1926, 1929, and 1930 were epidemic years. Nineteen twelve, 1915, 1926, and 1930 were years of low precipitation and nearly normal temperature years. The year 1924 had an increase in precipitation and a temperature slightly less than normal. Nineteen twenty-nine was a year of approximately average precipitation and temperature, while 1910 showed clearly increased precipitation and normal temperature. On the other hand, 1911, 1913, 1914, 1918, 1919, 1921, 1922, and 1923 were years of low precipitation and approximately normal temperature with no increase in the number of cases.

Burlington, Vermont. Nineteen fourteen, 1921, and 1923 could be considered epidemic years. Nineteen fourteen was a year of low precipitation and low temperature, 1921 a year of low precipitation and high temperature, while 1923 was a year of normal precipitation and slightly less than normal temperature. Nineteen fifteen, 1917, 1919, 1926, and 1927 were all years of low precipitation and low temperature, while 1928 was a year of low precipitation and normal temperature.

New York, New York. Nineteen twelve, 1916, 1921, 1923, 1927, and 1928 were epidemic years with 500 or more cases occurring during each of these years. Nineteen twelve, 1921, and 1923 were low precipitation, normal temperature years. The great epidemic year of 1916 was a year of average precipitation and nearly average temperature, though January and August of that year were "record" dry months. Nineteen twenty-seven was about average, while 1928 was wet though with an average temperature. The years 1911, 1913, 1914, 1925, 1929, and 1930 showed low precipitation and approximately average temperature with almost the ordinary or slightly increased number of cases.

Syracuse, New York. Nineteen sixteen, 1922, 1924, 1926, and 1930 were epidemic years. Nineteen sixteen was a year of low precipitation and about average temperature. Nineteen twenty-two was exceedingly rainy and about average in temperature. Temperature and precipitation readings were a little low in both 1924 and 1926, while in 1930 they were about average. Nineteen twenty was very dry with a slightly lessened temperature, but not an epidemic year. In 1923 and 1927, the precipitation readings were a little low though nearly normal in

TABLE I

MORBIDITY RATES AND CLIMATIC CONDITIONS

YEAR	NORTHEASTERN CITIES														
	BUFFALO			BURLINGTON			NEW YORK			SYRACUSE			CINCINNATI		
	CASES	PPT.	TEMP.	CASES	PPT.	TEMP.	CASES	PPT.	TEMP.	CASES	PPT.	TEMP.	CASES	PPT.	TEMP.
1910	52	1359	2323										0	1158	2618
1911	9	959	2411				358	1015	2569				102	1442	2739
1912	316	913	2298				504	1203	2521				8	1753	2712
1913	17	898	2313				310	1228	2554				7	1080	2746
1914	9	993	2276	31	987	2240	129	1160	2529	4	1436	2352	0	1010	2761
1915	31	754	2297	2	1010	2297	95	1359	2502	0	1365	2351	8	1580	2614
1916	13	1319	2327	4	1253	2293	9023	1415	2449	229	1192	2370	47	1311	2621
1917	8	1507	2175	1	999	2104	139	1717	2428	5	1218	2249	10	1569	2508
1918	7	906	2310	1	1406	2302	134	1488	2529	0	1252	2388	23	1646	2625
1919	3	959	2393	1	907	2307	42	1652	2535	0	1574	2425	2	1137	2650
1920	1	1104	2231	0	1643	2242	154	1837	2456	1	874	2308	4	1601	2520
1921	14	867	2509	9	799	2443	588	1368	2619	16	1344	2553	10	1461	2759
1922	4	947	2401	1	1650	2335	224	1961	2579	50	2462	2421	5	1063	2715
1923	11	958	2276	7	1253	2211	577	1019	2530	8	1053	2328	2	1099	2605
1924	34	1246	2194	0	1222	2212	344	1380	2437	152	1285	2271	9	1441	2648
1925	40	734	2273	2	1508	2251	307	1252	2551	5	1508	2344	7	1591	2675
1926	144	901	2116	0	1077	2141	111	1415	2425	66	1010	2230	1	2227	2548
1927	14	1328	2237	0	927	2234	537	1539	2445	2	1137	2297	69	1675	2593
1928	27	1564	2224	0	1099	2358	572	2043	2475	25	1475	2321	9	1810	2538
1929	102	1109	2291	1	1715	2288	71	1268	2553	24	1402	2429	18	2124	2610
1930	75	779	2300	0	1426	2327	78	1255	2570	150	1347	2477	50	650	2735
Average		1150	2306		1222	2339		1420	2513		1302	2486		1379	2618

Table II

Morbidity Rates and Climatic Conditions

Year	Middle Eastern Starboard Cities						Northern and Middle Western Cities								
	Baltimore			Philadelphia			Des Moines			St. Paul			Sioux City		
	Cases	Ppt.	Temp.	Cases	Ppt.	Temp.	Cases	Ppt.	Temp.	Cases	Ppt.	Temp.	Cases	Ppt.	Temp.
1910				155	1413	2672									
1911				63	1465	2677									
1912	25	1429	2670	45	1430	2637									
1913	2	1237	2710	24	1785	2686									
1914	1	1191	2710	18	1578	2638									
1915	26	1301	2690	12	1682	2638									
1916	206	1754	2670	1006	1195	2608									
1917	2	1659	2630	19	1331	2581	15	1920	2486	80	1346	2413			
1918	38	1399	2680	16	1584	2644	1	1549	2623	6	1348	2322			
1919	83	1941	2690	7	2021	2656	2	1830	2623	4	1332	2396			
1920	15	2033	2630	22	1635	2586	22	1414	2514	4	1798	2461			
1921	148	1548	2770	52	1217	2729	6	1449	2741	9	1329	2360			
1922	26	1661	2730	22	1097	2695				36	1193	2604			
1923	11	1015	2720	18	1048	2662				2	1096	2448			
1924	31	1922	2590	21	1777	2533	0	1227	2611	6	1127	2475	1	1524	2555
1925	28	1110	2730	16	1094	2707	6	1232	2489	17	1376	2250	0	1097	2418
1926	28	1226	2630	16	1533	2571	11	1106	2761	28	1360	2431	0	1097	2647
1927	11	1224	2650	53	1339	2569	11	1273	2573	3	847	2410	0	907	2593
1928	127	1732	2650	11	1831	2602	0	1113	2537	8	1084	2307	5	1683	2490
1929	9	1446	2710	45	1299	2676	7	1688	2512	20	1084	2329	1	1248	2475
1930	21	1094	2780	63	1391	2718	4	1271	2565	2	1277	2376	3	2646	2535
							27	996	2653	9	958	2469	39	1427	2626
Average		1508	2682		1370	2626		1561	2580		1335	2427		1361	2529

TABLE III

MORBIDITY RATES AND CLIMATIC CONDITIONS

YEAR	FAR WESTERN CITIES												MIDDLE SOUTHWESTERN CITY		
	FRESNO			LOS ANGELES			SACRAMENTO			SAN FRANCISCO			FORT WORTH		
	CASES	PPT.	TEMP.	CASES	PPT.	TEMP.	CASES	PPT.	TEMP.	CASES	PPT.	TEMP.	CASES	PPT.	TEMP.
1910															
1911															
1912										5	120	2206			
1913										16	426	2283			
1914										4	132	2308			
1915										13	167	2280			
1916										9	380	2338			
1917										31	10	2308			
1918										6	39	2275			
1919										16	60	2308			
1920										3	11	2280			
1921										3	140	2297			
1922							21	119	2660	60	106	2306			
1923							0	83	2590	1	128	2317			
1924							5	304	2600	4	404	2313			
1925	1	274		160	404	2490	0	36	2720	10	30	2363			
1926	4	411		35	793	2580	16	377	2690	66	686	2379	3	1297	3168
1927	17	86		142	277	2460	3	461	2800	4	541	2429	2	1497	2844
1928	3	29		45	258	2520	48	225	2630	90	243	2322	57	896	3097
1929	2	98		36	465	2480	4	84	2700	8	157	2359	2	2529	2982
1930	4	74		449	518	2550	1	150	2630	10	188	2395	3	852	3052
							11			230	182	2347	14	1498	3090
Average		145			433	2459		238	2630		237	2297		1463	3008

temperature. Nineteen twenty-two was a year of marked precipitation, over fifteen inches of rain falling in June alone, and yet the number of poliomyelitis cases was increased.

Baltimore, Maryland. Nineteen sixteen, 1919, 1921, 1924, and 1928 were marked epidemic years, but all were years of normal or high precipitation and average temperature. Conversely, 1913, 1914, 1915, 1918, 1923, 1925, 1926, and 1927 were years of low precipitation and practically normal temperature, with no case increase.

Philadelphia, Pennsylvania. This city had 2 epidemic years, 1910 and 1916. The precipitation and temperature of 1919 were above normal, while both were below in 1916. The years of 1922, 1923, and 1925 showed low precipitation and high temperature and theoretically should have been epidemic years.

Cincinnati, Ohio. Nineteen eleven, 1916, 1918, 1927, and 1930 had case increases. Nineteen eleven and 1916 were above normal in both precipitation and temperature. Nineteen twenty-seven was above normal in precipitation and about normal in temperature. Nineteen thirty was always below in precipitation with a slight increase in the temperature range. On the other hand, the years 1913, 1914, 1919, 1922, and 1923 showed decreased precipitation and slightly increased temperature and no morbidity increase.

Fort Worth, Texas. Nineteen twenty-seven and perhaps 1930 could be considered as epidemic years. The former had a definite decrease in precipitation with an average temperature. The latter was above normal in both readings. Nineteen twenty-nine, however, with only 3 cases, had a precipitation even less than 1927.

Des Moines, Iowa. Nineteen seventeen, 1920, and 1930 were epidemic years. Nineteen seventeen had greater precipitation than normal and a slightly subnormal temperature. Nineteen twenty-two was slightly under normal in both readings while 1930 was definitely below as far as precipitation was concerned and a little above in temperature readings. Nineteen twenty-one, 1923, 1924, 1925, 1926, 1927, and 1929 had lessened precipitation and normal or slightly above normal temperatures without any marked increase in cases.

St. Paul, Minnesota. Actually, 1916 was the only epidemic year but 1921, 1924, 1925, and 1928 had more cases than normal. Nineteen sixteen was a year of normal precipitation and temperature; the 1921 readings were lower than normal in precipitation but higher than normal in temperature; those of 1924 were normal in precipitation but below normal in temperature and in 1925, both readings were normal, while in 1928 there was a slight decrease in both precipitation and temperature. The precipitation readings in 1922, 1923, 1926, and 1930 were below average and the temperature readings about normal, while in 1927 both readings were below normal with no increase in the case morbidity rate.

Sioux City, Iowa. Only 1930 could be considered an epidemic year and here the precipitation and temperature were above normal. Nineteen twenty-four, 1925, and 1926 were years of low precipitation, but with no increase in cases.

Fresno, California. Nineteen twenty-five and 1927 were epidemic years, the first with a greater than normal and the latter with a decreased precipitation. Nineteen twenty-eight, 1929, and 1930 had less rainfall than normal with no increase in the number of cases. The temperature readings for this city were not included in the annual meteorologic summary.

Los Angeles, California. Nineteen twenty-five, 1927, and 1930 were epidemic years. In 1925, the temperature and precipitation were about average. In 1927, the precipitation was below and the temperature a little under normal. In 1930, although there was a slight increase in precipitation due chiefly to increased March

rains and a slight increase in temperature, this was the year of the greatest case morbidity. On the other hand, in 1928, the precipitation was below normal and the temperature nearly normal with no marked case increase.

San Francisco, California. Here the lack of connection between low precipitation and increased temperature is so obvious as to warrant no comment.

Sacramento, California. Nineteen twenty-one, 1925, and 1927 could be considered epidemic years for a city of this size. Nineteen twenty-one was low in precipitation and about normal in temperature. Nineteen twenty-five was above normal in both factors, while 1927 was approximately normal in both. The years 1922, 1924, 1928, and 1929 with no case increases were below normal in precipitation with an average or slightly increased temperature.

CONCLUSION

There is no connection between the changes in temperature and precipitation and the increase or decrease in the morbidity rate of poliomyelitis of the cities studied.

REFERENCES

1. Toomey, John A., and Lipson, Harry A.: The Seasonal Incidence of Poliomyelitis in and about Cleveland (in print).
2. Lavinder, C. H., Freeman, A. W., and Frost, W. H.: Public Health Bulletin No. 91, July, 1918.
3. Frost, W. H.: Hygienic Laboratory Bulletin No. 90, October, 1913.

INTRAUTERINE PNEUMONIA WITH EVIDENCE OF HEALING

Report of a Case

Edward Dyer Anderson, M.D., and John F. Pohl, M.D.
Minneapolis, Minn.

RELIABLE autopsy reports would indicate that pneumonia is a comparatively common finding in infants dying soon after birth.[1, 2] In some of these cases it is thought that the pneumonia was present before birth. Besides those cases in which death occurs after birth, there are many instances, where the baby is born dead, in which pneumonia has been shown to be present, indicating a fetal infection.[2] There is, however, evidence that many fetal infections have a direct relationship with the length of time of labor,[2, 3, 4] pneumonia being a more common finding after prolonged, dry labors, which would indicate that many so-called antenatal pneumonias are more specifically intranatal infections. There are, nevertheless, many instances of true fetal pneumonia. In reviewing the available autopsy reports of congenital pneumonia, one notices that the characteristic microscopic examination of the diseased lung reveals a few cellular elements, a variable number of red cells, an absence or a small amount of fibrin and slight involvement of the interstitial framework of the lung[2]—in other words, the histopathologic picture of a condition which in an older child would probably be regarded as a very early pneumonia or that resulting from an overwhelming infection. This has led to the conclusion either that the pneumonia occurred just before birth, or that the lack of inflammatory response is evidence of a poor defensive reaction on the part of the fetal tissues.[2, 5] In a review of the literature we have not been able to find any autopsy report of fetal pneumonia where there is unmistakable evidence of ability of the fetus to respond to the infection by showing organization of the inflammatory exudate and fibrosis of the lung tissue. Our reason for presenting this case is that the microscopic examination of the lung tissue showed a pneumonia with definite organization and fibrosis which would indicate that fetal pulmonary tissue may react defensively toward an infection in the same manner as is possible after birth.

Mrs. R. F. T., aged twenty-three years, had been married three years. She and her husband had always been in good general health. One year after her marriage she had a miscarriage with a two months' pregnancy. The course of her second pregnancy was uneventful except for one illness, and she came to term in very good physical condition. At the fifth month of gestation she had an attack of

Submitted for publication, March 29, 1932.

pain in her chest, associated with a bronchial cough. She thought she had no fever and did not consider the affection of sufficient severity to go to bed or to consult her physician. The attack lasted only a few days. She was examined at intervals by her obstetrician and was found to have no physical disorders during the pregnancy. The blood pressure and urinary findings were normal and the blood Wassermann was negative. On November 6, 1931, after an uncomplicated twelve-hour labor, she was delivered of an apparently normal full-term, living female infant. The postpartum course of the mother was uneventful and afebrile. The baby appeared normal at birth; respiration was spontaneous and circulation good. Twelve hours after birth the child began to have cyanotic intervals accompanied by difficulty in respiration. Examination at this time revealed a rapid respiratory rate; the transmission of breath sounds was diminished over the entire chest, but there was no impairment of percussion note. The chest findings were regarded as those of fetal atelectasis. On the second day, fine crepitant râles were

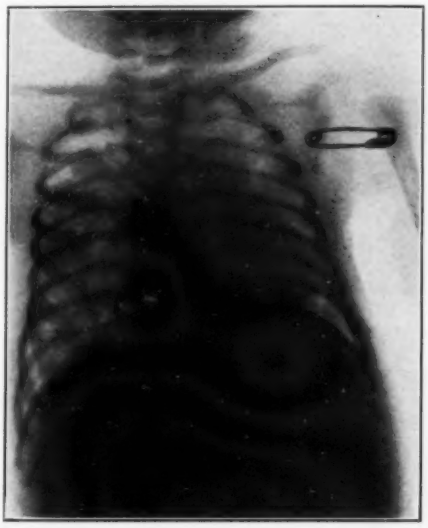

Fig. 1.—X-ray picture of chest, showing mottled densities particularly of the right side, representing atelectasis with some associated pneumonia.

heard over the right chest, but there was no change in the character of the breath sounds. Roentgenographic examination of the chest showed considerable atelectasis of both lungs, especially the right, with the appearance of an associated bronchopneumonia. The attacks of cyanosis became more frequent and were unrelieved by carbon-dioxide-oxygen inhalation to any extent. The child died on the fourth day, in asphyxia. The temperature had remained normal except for a terminal rise. The necropsy disclosed the atelectasis and pneumonia which had been diagnosed clinically and by x-ray examination. The microscopic examination showed an extensive fibrosis of the lungs which has been interpreted as evidence of an intrauterine pneumonic infection in the process of healing. Tissue stains for spirochetes were negative. No examination was made of placental tissue.

Pathologic Report.—Dr. E. T. Bell, and Dr. I. H. Kass, University of Minnesota. The gross and microscopic examinations of tissues except for the lungs, showed no pathologic condition. The right lung weighed 25 grams, the left 12.5 grams.

The right lung felt solid throughout, more marked in the two upper lobes. Throughout the posterior portions of both lungs there was a definite lumpy sensation. The surface of the pleura appeared normal. Both lungs cut with increased resistance, and the cut surface varied from a dull red color, seen especially in the right lung, to deep red hemorrhagic areas, noted particularly in the lower and posterior portions of both lungs. There were multiple small abscesses present throughout both

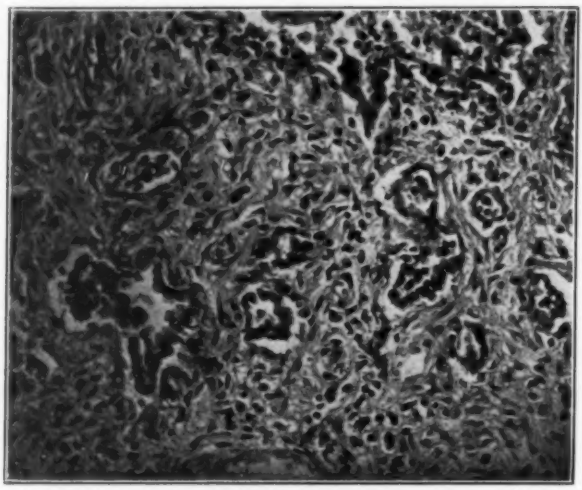

Fig. 2.

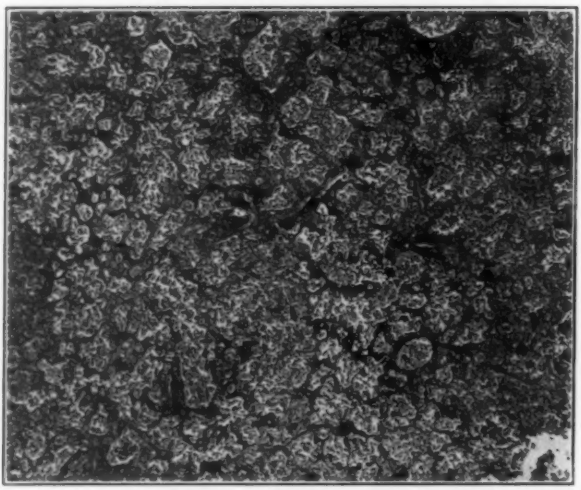

Fig. 3.

Figs. 2 and 3.—Low power and high power magnification showing the extensive organization and fibrosis of lung tissue with compression and obliteration of the alveoli.

lungs, varying from pinhead size to 3 mm. in diameter. Pressure on the cut surfaces of both lungs expressed a thin greenish-yellow pus from the abscess areas. The right upper and middle lobes had a firm, fibrous texture. Both lungs floated in water.

MICROSCOPIC EXAMINATION

Sections from some areas in the lungs showed a normal structure; in other areas the alveoli were filled with polymorphonuclear leucocytes and macrophages. There was no special relation of these areas of consolidation to the bronchi. Extensive areas of lung tissue in the right upper and middle lobes showed a much more striking change, the lung tissue being replaced largely by dense fibrous tissue. The alveoli appeared as small cysts lined with epithelium and separated by dense bands of collagenous fibers. The fibrous interalveolar septums occupied a much greater area than the alveolar spaces. The dense fibrous septums established the fact that the inflammation was of long standing.

Diagnosis.—(1) Acute interstitial pneumonia; (2) pulmonary atelectasis; (3) multiple abscesses of both lungs; (4) bronchopneumonia.

REFERENCES

1. Cruickshank, J. N.: M. Res. Council, Special Report Series No. 145, 1930.
2. Johnson, William C., and Meyer, Juvenal, R.: Am. J. Obst. & Gynec. 9: 151, 1925.
3. Kobak, Alfred J.: Fetal Bacteremia, Am. J. Obst. & Gynec. 19: 299, 1930.
4. Browne, Francis J.: Brit. M. J. 1: 469, 1922.
5. Gordon, Abraham S., and Lederer, Max: Am. J. Dis. Child. 36: 764, 1928.

87 S. Seventh Street.

ANTIQUES OF PEDIATRIC INTEREST

T. G. H. Drake, M.B., F.R.C.P.(C)

THE two end examples are typical blood porringers bearing graduations on the interior. The center specimen is of the barber surgeon type with a depression for fitting to the area to be bled, or for holding under the chin when used for shaving. Brass vessels of this shape still act as signs outside the barber shops in Copenhagen. Great care must be exercised in purchasing pewter bleeding bowls, since there are many specimens cast from the old moulds on the market and in addition many antique plain porringers have been engraved with graduations recently.

Bleeding bowls, pewter

Left, English, circa 1800, capacity 16 ounces; German, circa 1780, 6 x 8 inches; French, circa 1800, capacity 500 gmes.

Among the ancients, blood-letting in childhood was usually frowned upon "Since in those times, physicians never attempted to take away blood in those fevers which were attended with any eruptions, particularly in children who are the most liable to this kind of illness. Their reason was, lest that emptying of the vessels should hinder the coming out of what was to be discharged by the skin" (Mead, 1755). However, Mead, who observed that "it is always better to try a doubtful remedy than none," on being consulted by another physician concerning what method he used in curing the measles, as none of his patients died of that disease, candidly answered, that he always or-

From the Department of Pediatrics, University of Toronto, and the Hospital for Sick Children, Toronto.

dered bleeding. In smallpox, while "many of our physicians imagine that they have done their duty when they have ordered one bleeding," Mead stated that "this is certainly an ill-timed caution; for in youths and adults it is often necessary to draw blood two or three times, only with an intermission of two or three days between each time."

Sydenham, 1722, in a peripneumonia following measles, "I have ordered the smallest infants to be bled in the arm, and have taken away that quantity of blood which their age and strength indicated with very great success. Nor is there any reason why one should wonder that I bleed young children, since from what I have hitherto observ'd it may be as safely perform'd in children as in grown persons. For by what other means can we relieve those that are breeding teeth from convulsions. I pass by at present the wonderful relief which bleeding gives in Hooping Cough, wherein it far surpasses all pectoral medicines whatever."

Von Rosenstein, 1776, recommended blood-letting in renal colic, in measles, in smallpox, in dyphtheria, and in convulsions. He stated that in pneumonia complicating measles the bleeding should be on that side where the cheek looks reddest; in pleurisy, from the arm of the affected side.

Mead, in a fifteen-year-old boy suffering from smallpox, performed six blood-lettings, each of five ounces. Evanson and Maunsell, 1842, give as the amount of blood to be withdrawn at any one time, "During the first year, from an ounce to three ounces of blood, will be sufficient; and after the first year, we may usually take an additional ounce for each year of the child's age, seldom having occasion within the period of childhood to exceed eight ounces of blood. As a general rule, it is well to stop the flow of blood when decided pallor takes place, without waiting for actual fainting."

Critical Review

NEUROLOGIC CONDITIONS IN INFANTS AND CHILDREN

R. Cannon Eley, M.D., Boston

SUBDURAL HEMATOMA IN INFANTS

THE etiology of subdural hematoma which more commonly has been called pachymeningitis hemorrhagica interna, continues to be questionable. Trauma and infection are the two causative factors that have received more credence than others.

As pointed out by Sherwood,[1] Hunt,[2] Peet and Kahn[3] convulsions or gradual enlargement of the head are the two most common symptoms and usually are noted before optic atrophy, retinal hemorrhages, or downward displacement of the eyes. In many instances the occurrence of an initial, severe, and persistent convulsion may be the mode of onset. As increased intracranial pressure may be present in infants for several weeks without presenting clinical manifestations,[4] it is not unusual for the infants to present the clinical appearance of hydrocephalus before the disease is suspected. Once suspected, the diagnosis can usually be established by unilateral or bilateral subdural punctures which will yield the typical grossly bloody or xanthochromic spinal fluid.

While the treatment of subdural hematoma is unsatisfactory at its best, and although Bagley[5] has presented experimental evidence which suggests that the presence of a very small amount of blood may produce changes of a serious nature, yet in the cases that are diagnosed early, repeated drainage of the cerebral subdural area may prove helpful. Peet and Kahn[3] have pointed out that the operative risk is great and that few infants are able to withstand the procedure. This is clearly shown in this statement which appeared in their recent communication: "The dura was opened exposing a gelatinous appearing cyst wall covering the entire brain. The cyst was opened, considerable hemorrhagic fluid escaping. The tough false membrane over the arachnoid was removed as far as possible, as well as the gelatinous lamination adherent to the dura. The condition became poor and the child died during the closure." Bagley,[6] in treating a comparable condition occurring in young adults has shown that satisfactory results may be expected in properly selected cases. With further study it is probable that similar results may be obtained in the treatment of infants.

Prognosis in this disturbance is poor.[7, 8, 9] In Sherwood's group three became feebleminded, one died from an intercurrent infection (this is the most common cause of death), one still had an active process after seven months, and four had not been followed long enough to warrant any conclusions. In Hunt's group four were feebleminded, one died, and one showed no sequelae after nine months.

From the Infants' and Children's Hospitals and the Department of Pediatrics, Harvard Medical School, Boston.

BIRTH INJURIES

Injuries to the central nervous system sustained at the time of birth may involve the peripheral nerves, the spinal cord or the brain and its appendages. The diagnosis of spinal cord or peripheral nerve injury is not difficult, whereas intracranial injury may easily be confused with other conditions. Hemorrhagic diseases of the newly born, atelectasis, either primary or secondary, prematurity, congenital heart disease, developmental defects of the central nervous system and prematurity,[10] all offer difficult differential diagnostic problems. Recently Goldzieher and Gordon[11] have called attention to the similarity of the clinical manifestations of adrenal hemorrhage and intracranial hemorrhage in the newborn. Clifford and Hertig[12] in their discussion of erythroblastosis fetalis, point out that the cyanosis, dyspnea and muscular twitchings seen in this disturbance may simulate intracranial hemorrhage. Munro[13] in the study of a large series of intracranial injuries in the newborn has called attention to the various pitfalls encountered in differentiating between such conditions as fracture of the skull, edema, and intracranial hemorrhage. Grulee[14] in a recent publication has also pointed out the dangers involved in establishing this diagnosis and in the conclusion of his communication states, "Primarily we are at present in no position to make a diagnosis of intracranial hemorrhage in the newborn in the majority of instances. Many of the cases which are now diagnosed hemorrhage are unquestionably due to other conditions. Information received from lumbar puncture is by no means always reliable, and must be subjected to very critical judgment. The reason for this lies primarily in the failure of physicians to realize the difference in symptomatology between the newborn and the older infant. It also lies in the lack of study of this group of conditions at this age, and in complacence with our present knowledge which would not be tolerated at any other period of life."

The various factors involved in the production of intracranial injury are not clearly understood, although trauma associated with the mechanisms of birth takes precedence. Rainey[15] found 350 injuries in 8,000 infants delivered alive at the Royal Maternity Hospital at Glasgow. Gillespie[16] in a study of the causes of death in 338 cases found that 29 to 88 per cent of all stillbirths were dependent on trauma. Ehrenfest[17] in his report on birth injury in its entirety makes the following statement, "A factor of outstanding significance in intranatal and neonatal mortality and infant morbidity is the traumatization of the child incident to birth. An increasing appreciation of this source of loss in infants' lives during or soon after delivery is unmistakably expressed in the gradual and steady increase in the number of death certificates that give birth injury as the cause of death. Even so, official vital statistics are still far from disclosing actual conditions."

Irving[18] in a study of the obstetrical aspect of intracranial hemorrhage of the newborn at the Boston Lying-In Hospital obtained the following statistics: From 1920 to 1930 there were approximately 13,849 deliveries. A clinical diagnosis of death from intracranial hemorrhage was made in 99 cases. From 1909 to 1929, 182 autopsies were made which included an examination of the head, and intracranial hemorrhage was found in 73 cases or 40 per cent. In regard to the frequency of this condition in relation to the type of delivery his figures show: Cesarean section 0.3 per cent, normal delivery 0.4 per cent, low forceps 0.5 per cent, midforceps 3.0 per cent, high forceps and breech extraction 2.6 per

cent, and version 1.7 per cent. Of the 73 necropsy studies that showed intracranial hemorrhage, hemorrhage occurred in 23 that were delivered normally, in 8 delivered by midforceps, in 7 delivered by high forceps, in 6 delivered by low forceps, in 3 delivered by version, and in 3 delivered by cesarean section. These figures, as Irving states, "indicate the greatly increased risk encountered by babies who were subjected to the more difficult methods of delivery." They also show that neither normal deliveries nor cesarean sections are free from danger. Eden[19] has called attention to the high percentage of intracranial hemorrhage occurring in infants delivered by cesarean section and has suggested that it might be due to the unavoidable compression of the infant's head in withdrawing it through the uterine incision. If the delivery was performed prior to the onset of labor, and if there was no evidence of intrauterine asphyxia, his explanation is plausible. However, if the operative procedure had been necessitated by failure of progress or by asphyxia, it is not inconceivable that the hemorrhage was secondary and not the result of the delivery. As regards cesarean section, Clifford[20] has made the following interesting observations: In the years 1930 and 1931, there were 64 cases of intracranial hemorrhage occurring in normal deliveries at the Boston Lying-In Hospital and in this group there were 3 cesarean sections. Two were performed on account of placenta previa and neonatal asphyxia. Both infants showed diffuse *petechial hemorrhages* at necropsy. The third was performed after the patient had been in labor for 48 hours, and 7 hours after the membranes had ruptured. At necropsy there was *gross* intracranial hemorrhage. In this instance the hemorrhage was probably the result of constant pressure of the fetal head against the pelvic bones. In 128 premature births there was a gross mortality rate of 25.7 per cent, and of this group, those delivered by cesarean section had a fatality of 44.5 per cent, those by low forceps 10 per cent; bag, version and extraction 100 per cent; breech 36.8 per cent; and normal deliveries 18.9 per cent. The high percentage of premature infants showing intracranial hemorrhage, even when delivered by cesarean section, is worthy of note.

During recent years attention has been directed to the importance of intrauterine asphyxia either as a direct factor or as a predisposing factor in the production of intracranial hemorrhage in the newborn, and although not conclusive, yet sufficient data has been collected to suggest that asphyxia may play an important rôle. Intrauterine asphyxia may occur as the result of a variety of conditions, but it more frequently occurs as a result of interference with the fetal circulation either by pressure or by coiling of the cord, by prolonged pressure of the fetal head on the pelvis, cervix, or perineum, or by alterations in the placental site. However, the exact mechanism by means of which hemorrhage may result from asphyxia is not clearly understood. Yagi[21] in his careful studies of this condition has suggested that it might be due to a combination of any two of the following three factors; i.e., stasis, pressure, or resistance of vessels. For example, if the pressure is prolonged, stasis (asphyxia is secondary) develops and finally the resistance of the blood vessels is overcome and hemorrhage occurs. Necropsy studies on infants dying from hemorrhage considered as secondary to asphyxia show petechial hemorrhages throughout the brain (and viscera) in contrast to the gross hemorrhage encountered when trauma is the etiologic factor.

The clinical manifestations of intracranial hemorrhage may appear immediately after birth, or years may elapse before the sequelae are observed. As Crothers[22] has pointed out, the injuries to be considered are, gross meningeal hemorrhage due to rupture of the tentorium or falx cerebri, intraventricular hemorrhage into the substance of the brain, and the effect of prolonged pressure without gross injury. In severe subtentorial hemorrhages the infant frequently dies from medullary injury or medullary compression. Supratentorial hemorrhages are compatible with life unless there is a rupture of the tentorium with resulting propagation of pressure.

Although there are opinions to the contrary, yet it is commonly held that cerebral palsies, idiocy, and mental retardation are frequent sequelae of intracranial hemorrhage. Ford,[23] however, states that only about 6 per cent of all cerebral palsies are due to birth injuries. "The common diffuse meningeal hemorrhage which is not large enough to cause death, apparently leaves no residium in the overwhelming majority of cases. The real birth injuries to the brain are caused by the rarer intracerebral hemorrhages and necroses, by depressed fractures with laceration of the brain, and undoubtedly by some more or less encapsulated meningeal hematomas which compress and soften the cortex." He also feels that the diplegias which constitute the largest group of cerebral palsies, are "the result of physiologic processes of intrauterine origin," and not due to meningeal hemorrhage at birth. Shroeder[24] in a study of the mental development of children with intracranial hemorrhage concluded that the mental retardation was not related to the presence of palsy. Winkelman[25] believes that the danger of the development of mental retardation may be minimized by repeated drainage of the cerebrospinal fluid in cases where intracranial hemorrhage has occurred. In the study of the fixed lesions resulting from old intracranial injuries in infants and children, Crothers[26] has demonstrated the value of encephalography, and as a result of this work, a clearer understanding of these patients has been made possible.

Injuries involving the various levels of the spinal cord and the brachial plexus have been so completely discussed by Crothers and Putnam[27] that the reader is referred to their original communication. More recently Byers[28] has described in detail not only the extensive and complex reflex activities that may be carried on in the isolated lower segment of a transected spinal cord, but has also compared the pathologic condition of the transected cord with the normal cord of an infant of the same age.

ENCEPHALITIS

Whooping Cough Encephalitis.—During the past year little, if any, literature dealing with encephalitis as a complication of whooping cough appeared, although in the years just preceding 1931 there were several communications. As the clinical manifestations do not materially differ from those seen in the other forms of encephalitis, these communications were largely concerned with the complications and with the postmortem studies of the central nervous system. In 1924 Husler and Spatz[29] reported two cases of whooping cough encephalitis, one dying three days after the onset of convulsions, and the other three weeks after the onset from a terminal pneumonia. Autopsy examination showed no gross

hemorrhage; microscopic examination of the cortex, dentate nucleus, and corpus striatum revealed definite cellular changes with unquestionable death of the cells and proliferation of the neuroglia. These observations were later confirmed by Jochims,[30] Ford,[31] and others.[32] Singer[33] has reported six cases that showed a diffuse hemorrhagic encephalitis and stated that this feature was of diagnostic importance in differentiating whooping cough encephalitis from the toxic and epidemic forms. From these observations it at once becomes obvious that gross hemorrhage, although it may occur,[34] is not, as previously considered, the most common cause of convulsions occurring in this disease. In view of the fact that convulsions are of prognostic importance one should always exclude other etiologic factors which may precipitate this symptom, for example, tetany.[35, 36] Sequelae such as blindness, spastic paralysis, mental retardation, epilepsy, and cerebral atrophy are not uncommon complications of this form of encephalitis.[37]

Mumps Encephalitis.—The neurologic complications of mumps may vary from a localized neuritis, for example, unilateral facial paralysis, to meningitis or encephalitis; the latter, however, is the more common complication. The etiology is not clearly understood, although the experimental work of Gordon[38] would indicate that a filtrable virus is probably the etiologic agent. The time of the appearance of these symptoms is quite variable as they may appear several days before the parotid swelling, they may accompany the parotitis, or they may appear several days after the swelling has subsided. Equally as variable are the symptoms, for in some cases they may consist of only headache, nausea and vomiting,[39] while in others, stupor, coma, retraction of the neck, and an exaggeration of the deep reflexes may be present.[40] Examination of the cerebrospinal fluid shows the change usually found in any of the various encephalitic conditions and is not of diagnostic value for mumps meningitis alone. Treatment should be directed along those lines that will relieve the increased intracranial pressure and of these therapeutic procedures, lumbar puncture with removal of fluid is the most valuable.[41] Rolleston[42] has emphasized the importance of keeping these patients confined to their beds for a period of three weeks. The prognosis is usually good although the condition may occasionally prove fatal.

Chickenpox Encephalitis.—Encephalitic manifestations occurring in the course of chickenpox are not common, and usually are not severe. Clinically these symptoms are observed between the fourth and fourteenth day after the appearance of the eruption and are accompanied by muscular weakness and paralysis. Although the lower extremities are more commonly involved, yet ocular palsy, dysphagia or paralysis of other groups of muscles may occur. The temperature is rarely above 100° F., and stupor and mental dullness are as rare as delirium and irritability are common. Examination of the spinal fluid usually shows the presence of globulin and a lymphocytosis varying from 5 to 71 cells; cultures of the spinal fluid are uniformly sterile. One might say that the two most striking features of this disease are the mild course and the absence of sequelae.

Zimmermann and Yannet[43] collected 19 cases from the literature and described in detail the pathologic changes involving the central nervous system in one of their patients. As shown by the following quotation, the pathology is not characteristic: "Widespread, noncharacteristic

degenerative lesions of the ganglion cells with a perivascular destruction of the myelin sheaths in the white matter of the parietal and occipital lobes. There was an extensive phagocytosis of fat in these regions and collections of fat granules in the leptomeninges. Small focal hemorrhages were scattered in the cerebral cortex."

Measles Encephalitis.—Encephalitis occurring as a complication of measles is a well-recognized clinical entity. Ford[44] collected 113 cases from the literature and added 12 of his own. The onset of symptoms is usually on the fourth to the sixth day with drowsiness and convulsions followed by stupor. Muscular rigidity, and twitchings are typical of this stage. After two or three days the patient may recover completely or the stupor may be prolonged and be accompanied by a great variety of central nervous system manifestations. From his large series Ford distinguished the following clinical types: (1) A mild and transient disturbance associated with headache, stupor, and signs of meningeal irritation. The characteristic feature of this group is the absence of focal signs, and, as one might expect, recovery is usually prompt and complete. (2) Multiple focal or diffuse lesions involving the basal ganglia, cerebral cortex, brain-stem, cerebellum or spinal cord. In this group of forty-two cases complete recovery occurred in fourteen, residual mental defects were noted in fifteen, disturbances in motility in eleven, narcolepsy in one and epilepsy in four. (3) Single focal lesions. This group comprised thirty-seven patients. One patient died, eight made a complete recovery and twenty-eight had hemiplegia. (4) Cerebellar syndromes were noted in fourteen patients with two complete recoveries. (5) Spinal cord syndromes and paraplegia. In this group the clinical course is that of an ascending paralysis. There were six deaths and fifteen complete recoveries in a group of thirty-three patients. (6) Very rare complications of the central nervous system such as psychoses. Ford's statistics show complete recovery in 25 per cent, death in 10 per cent and residual symptoms in 65 per cent.

An unusual manifestation has been reported by Heath[45]; a boy on the eleventh day of an uncomplicated course of measles suddenly became blind and then developed the typical encephalitic picture. Opthalmoscopic examination at this time showed findings resembling those of bilateral arterial embolism, an observation not previously reported. During the course of his illness the patient's sight returned but definite personality changes remained.

Necropsy studies have shown rather constant abnormalities. Congestion and hemorrhages are the most common findings, although the perivascular demyelinization is the most characteristic change seen in the disease.[46, 47] Greenfield[46] has emphasized the marked similarity in the pathologic findings in postvaccinal encephalitis and in encephalomyelitis following measles, and considers that the observations support the theory "that both the postvaccinal and other forms of encephalitis which follow eruptive fevers are not directly due to the virus of the preceding fever, but to another virus which is either stimulated to activity or is directed against the nervous system by the exanthem." When one considers that the mortality rate in encephalomyelitis complicating measles is 10 per cent, whereas in postvaccinal encephalitis the mortality rate is 58 per cent, it is a little difficult to understand how the virus can be the same in the two diseases.

Postvaccinal Encephalitis.—Although occasional cases of postvaccinal encephalitis may have occurred in the past yet it was not until 1922 and 1923 that the attention of the medical profession in general was attracted to this complication of one of the oldest prophylactic measures in medicine. The impetus for this study was furnished by the sudden appearance of a large number of cases in both Holland and England, and although a few cases have been reported from other countries, yet the greatest number have occurred there.

Naturally the first question was that concerning the etiology—was the disease due to the activation of an already existing neurotrophic virus by vaccinia, or was it due to the virus of vaccinia? Troup and Hurst[48] in a review of the literature and a case report concluded that the pathologic changes were indistinguishable from those in disseminated encephalomyelitis following measles, vaccination, etc., and suggested that all of the cases were examples of a single disease which was awakened by the causative organism of the acute fever or by the lowering of the patient's resistance. McIntosh and Scarff[49] in 1930, concluded that virulent strains of vaccinia when injected intracerebrally, intradermally, or intravenously into rabbits produced a definite meningoencephalitis and that the lesions produced were comparable to those observed in postvaccinal and postvariola encephalitis in man. Eckstein[50] has recently reported the presence of the virus in the cerebrospinal fluid of five patients with central nervous system involvement following vaccination. The virus, although present in the blood stream of vaccinated individuals between the sixth and eighth day, is never present in the cerebrospinal fluid of individuals without encephalitis. Experimentally the author was only able to produce encephalitis in those monkeys in which the circulation of the cerebrospinal fluid had been disturbed such as by repeated lumbar puncture or ligation of the jugular vein. He has concluded that an individual constitutional factor is present in those patients who develop central nervous system involvement and cites the incidences of two or more cases in the same family. In a recent review of this form of encephalitis and allied conditions Jorge[51] has discussed both views concerning the etiology and is of the opinion that the evidence is not yet conclusive.

Little has been added to the symptomatology of the disease. In brief, the cardinal symptoms as given in the excellent communications of Flexner,[52] Jorge,[51] and Thompson[53] are as follows: abrupt onset with headache, vomiting, pyrexia and paralysis; convulsions though common in infants, are not frequently encountered in children and adults. The temperature rises rapidly, profound prostration develops, and death may occur in from two to four days. Older children who escape these intense manifestations usually develop paralysis of one or more extremities. Physical examination presents an acutely ill patient with signs and symptoms referable to the central nervous system. Examination of the spinal fluid reveals an elevation of pressure but otherwise normal fluid except in the fatal cases where there is an increase in the protein and mononuclear cells. In Thompson's collected series of 298 cases 65 per cent became ill between the tenth and thirteenth day and 82 per cent between the seventh and fourteenth day following vaccination. Miller[54] has reported a case with the onset two days after vaccination. One of the outstanding features is the low morbidity and the high mortality. The Dutch mortality rate was 58 per cent and the English 35 per cent.

Flexner has suggested that this difference has been due to the inclusion of more mild cases in the English than in the Dutch statistics. Recovery, when it occurs, is usually complete.

The pathology presented by these patients is both constant and typical. The meninges show the least involvement as the characteristic lesions occur within the central nervous system and may extend from the cortex to the lumbo-sacral cord. Microscopically one finds degeneration, exudation and proliferation. Perivascular demyelinization is very prominent and may extend over large areas.

In view of the age incidence of postvaccinal encephalitis it is probably a good prophylactic procedure to vaccinate the children either before three years of age or after they are ten years old, as the majority of cases have occurred between these two age periods. In Germany and in the United States a great many children are vaccinated during the first two years of life and this may account for the low incidence in these countries. In Holland the greatest number of children are vaccinated at the time of their entry to public schools. Gordon,[55] Thompson,[53] Jorge,[51] and others have suggested the use of human blood serum (from a patient recently vaccinated) as a therapeutic measure in the treatment of the acute stage. This form of treatment has been employed in a few instances and at times has been followed by an immediate clinical improvement.

Lead Encephalitis.—The one form of encephalitis that has probably attracted more attention than any other during the past year, has been that associated with lead poisoning. Usually one associates plumbism with adults who employ lead in their work, but recent literature has shown that the disease is not uncommon among infants and children, in fact with the present diagnostic aids one might say that it is relatively common.

The occurrence of plumbism does not necessarily imply that the individual is suffering from encephalitic manifestations, although in McKhann's[56] report of forty cases of plumbism twenty-four had cerebral involvement. The probable explanation for this high percentage lies in the fact that lead poisoning is rarely suspected in infants and children, and therefore the patient may have been ingesting it over a long period of time without ill effect and then suddenly in the presence of some infection, a quantity sufficient to produce acute cerebral manifestations is liberated.

In infants the most common source of lead is from the paint on their toys, whereas in children the paint is usually ingested by chewing pencils, wooden penholders, crayons, and furniture. For this reason the disease is more commonly seen in children suffering from pica;[57] i.e., perversion of appetite. Other sources of lead are found in the use of lead nipple shields,[58] water from lead pipes, and occasionally face powder.

In the mild cases of plumbism the symptoms are referable to the gastrointestinal tract with anorexia, constipation, and abdominal pain, whereas in the more severe cases the symptoms are referable to the central nervous system with stupor, coma, convulsions, delirium, projectile vomiting and papilloedema. Associated with these symptoms there is a secondary anemia and an elevation in the arterial blood pressure. This elevation in blood pressure is considered as being secondary to the cerebral edema which is present, in varying degrees, in

patients with cerebral involvement. Not only has this edema been observed at necropsy but it has also been experimentally produced in animals.[59] The clinical course of the disease does not differ from that seen in the various other forms of encephalitis. The sequelae are usually severe as the patient is frequently left with blindness, epilepsy, generalized spasticity, or mental retardation. In McKhann's[56] twenty-four cases there were nine deaths, and five cases of "permanent injury presumably due to encephalitis."

The diagnosis is not without pitfalls and one should not depend on any one factor. A carefully taken history is probably the most important aid and if this is executed in minute detail one will usually be rewarded for his work. Physical examination yields the least information as the neurologic findings do not differ from those seen in other forms of encephalitis. The lead line, due to the deposit of lead sulphide in the capillaries at the gum margins, is rarely seen in children. Basophilic stippling of the erythrocytes, although common in plumbism, also occurs in other conditions, and may even be absent in patients who have large deposits of lead. The presence of small amounts of lead in the stools and urine is not sufficient for diagnosis as Kehoe and Thamann have shown that some degree of lead absorption is "normal" and that during disease it may appear in the urine and feces.[60] Perhaps one of the most helpful diagnostic procedures is the evidence obtained by the roentgenologic examination of the long bones. Park,[61] Vogt[62] and Caffey[63] have demonstrated the presence of a dense zone, or band, at the growing ends of the long bones and at the margins of the flat bones in infants and children suffering from lead poisoning. Necropsy studies have shown that this dense band may be the result of densely packed trabeculae[61] or to the deposit of lead.[62] In view of the close relationship existing between lead and calcium metabolism[64] care must be exercised in interpreting the roentgenograms. This is particularly true when dealing with patients who have been receiving small amounts of phosphorus.[65] The use of parathormone[66] may, at times, prove helpful in establishing the diagnosis although the possibility of liberating a large amount of lead is not without danger.

The treatment of lead encephalitis should be directed along two lines, first the removal of lead from the circulation and its fixation in the bones, and second the introduction of proper therapy for the reduction of cerebral edema. The administration of calcium lactate by mouth in conjunction with a diet high in calcium is thought to remove rapidly the lead from the circulation.[67] Calcium chloride by mouth should be avoided as this is an acid-producing salt[68] and would result in a liberation of lead, thus aggravating the condition. Cerebral edema can be relieved by appropriate measures.[56] Experience has shown that efforts at de-leading these patients are not to be advocated as they may cause an exacerbation. Furthermore, the patient will, in the course of time, gradually de-lead himself without danger.

In view of both the high mortality rate and high morbidity rate one should consider lead poisoning in any infant or child that presents evidence of encephalitis.

EPILEPSY

The present situation in the study of epilepsy may be summarized in the following quotation. "Fifty-six clinical causes of fits have been

grouped and discussed. Thirteen physiologic mechanisms have been postulated to explain the occurrence of these seizures. To simply further and relate seizures to one or two simple mechanisms will be the work of years of further research. At present one might tentatively reduce the number of mechanisms by saying that it seems probable that embryologic defect and tissue destruction act by altering neural conduction. Congestion, vasoconstriction, asphyxia, and increased intracranial pressure might all act by means of cerebral anoxemia. This includes 33 of the 56 causes given, and makes the largest group. Hydration and dehydration probably act through chemical changes (e.g., acid-base relationships), and the mechanical theory that they act by pressure is controverted. A large group of drugs and chemicals, apparently quite unrelated in other ways, cause convulsions by some entirely unknown mechanism. There is still much research to be done before one can explain the causes of a fit.''[69]

Fay[70] and McQuarrie[71] have done a great deal of work on the influence of water balance on the frequency of convulsive attacks. The former has offered the ''mechanical theory of epilepsy'' and is of the opinion that the predisposing factor is hydration. It is his belief that there is some pathologic condition of the arachnoid villi or pacchionian bodies which interferes with their function, thereby resulting not only in a damming up of cerebrospinal fluid within the subarachnoid spaces, but also in an increase in the supracortical pressure. As a result of this pressure a relative ischemia is produced which in turn may produce cortical atrophy. The chief objections to this theory, as pointed out by Cobb[69] and Fremont-Smith,[72] is that it is quite difficult to explain how the pressure in the subarachnoid spaces over the cortex could ever become greater than the pressure within the ventricles. McQuarrie although supporting the theory of hydration, has advanced the tentative explanation, not on a mechanical basis, but on disturbed physiologic function of the cells with resulting imbibition of an abnormal quantity of water. In a recent intensive study both on the mineral and water balances of a severely epileptic child McQuarrie[73] and his coworkers have shown that the establishment of a positive water balance was always followed by the occurrence of convulsive seizures. From twelve to thirty-six hours before the appearance of the seizures, and therefore during the period of positive water balance, there occurred a striking increase in the potassium to sodium ratio in the urine whereas during the periods of diuresis when no convulsions were occurring, sodium and chlorine were the predominating minerals in the urine. So consistent was this observation that McQuarrie has suggested that there might be some physiologic disturbance in the permeability of the cell membrane, particularly the cells of the central nervous system, which resulted in a loss of potassium and an increase in water content.

A detailed study on the causes of epilepsy by Dr. Stanley Cobb,[69] with discussions by Dr. Temple Fay, W. G. Spiller and E. Spiegel has recently been published in the *Archives of Neurology and Psychiatry.* For more complete discussion of the entire subject of epilepsy one may refer to the late publication of the Association for Research in Nervous and Mental Diseases, *Epilepsy and the Convulsive State,* Baltimore, Williams and Wilkins Co., 1932.

Treatment of epilepsy may be directed along several lines; i.e., by limitation of fluids or by diuresis thereby producing a state of dehydration or by establishing a state of ketosis by means of the ketogenic diet, or by the use of sedatives or by surgical intervention. Both Fay[74] and McQuarrie[71, 73] have shown that during dehydration the tendency to convulsive seizures is diminished. The ketogenic diet[75] which consists of limited amounts of carbohydrate and protein and a large amount of fat has proved successful both in the control of seizures, and in diminishing their frequency. Helmholz[76] and Talbot[77] have obtained complete relief of attacks in about 33 per cent of their patients and improvement in an equal number. Whether the good results obtained by this form of therapy are due to the ketosis or to the release of a large amount of water that is not longer necessary for carbohydrate metabolism and thereby establishing dehydration, is problematical. Peterman[78] advocates the following régime for the epileptic child (if he cannot be placed in a hospital for fasting and the ketogenic diet): (1) A definite daily regimen which includes a long nap, and a long sleep, meals at regular hours and a daily bowel movement; (2) a restricted carbohydrate and protein and a high fat diet; (3) fluid restriction to a minimum, at least to 600 or 800 c.c. daily and less if convulsions continue, and (4) phenobarbital in large enough doses to control seizures that may occur after or until the foregoing procedures are established." McClintic[79] has reported successful treatment of epilepsy by the surgical removal of the vasoconstrictor nerves to the cerebral vessels arising from the sympathetic ganglia of the upper thorax, neck and carotid bodies. This procedure presupposes that the epileptic attacks are due to angiospasm of the cerebral vessels. The best results that he has obtained in children have been in those under nine years of age and who had a clear cut history of birth injury. Foerster and Penfield[80] have obtained gratifying results in the surgical treatment of traumatic epilepsy, and have presented evidence to show that the displacement of the ventricular system in this group of patients is due to cicatricial contracture which gradually draws one or both hemispheres toward the lesion.

ENCEPHALOGRAPHY

The injection of air either directly into the ventricles or indirectly by means of the spinal subarachnoid route has been employed for several years as a diagnostic procedure in the study of patients suffering from disturbances in the secretion and absorption of cerebrospinal fluid, and from neoplasms.[81] However it is only within recent years that its value has been recognized both as a therapeutic agent[82, 83, 84] and as a means of more adequately studying infants and children suffering from a large variety of neurologic conditions[85]—particularly those related to fixed lesions of the brain.[86, 87]

The employment of this method in children is, however, somewhat different from its employment in adults. In the latter its greatest value lies in the localization of cerebral tumors, whereas in the former its value lies in the study of that large group of children suffering from what may be termed "fixed lesions." Crothers[86] in 1930 clearly demonstrated the value of this procedure in the study of children suffering from intracranial hemorrhage, developmental defects, epilepsy, etc. Not only can one more readily understand the clinical manifesta-

tions, but it also serves as rather convincing proof as to the futility of surgical intervention (a question that is foremost in the minds of the parents) in the majority of these patients. Furthermore the parents of these children, when confronted with the evidence of cerebral damage as demonstrated by air injection, will accept it as final and cease their pilgrimage from doctor to doctor. There are occasions when this alone justifies the procedure.

Experience has shown that the reactions[88] following encephalography may not only be severe but may even be fatal. This recently recognized diagnostic procedure should not be used except when there is a reasonable hope that the evidence secured will serve a useful diagnostic or therapeutic end. It should not be attempted by anyone who is not thoroughly familiar both with the technic involved and with the complications that may arise.

The proper interpretation of the roentgenogram is by no means a simple procedure. Films showing large dilated ventricles or large collections of air in the subarachnoid spaces obviously offer no difficulty. However, one frequently finds normal ventricles with only slight exaggeration of the cortical markings and herein lies the difficulty, for little is known as to the norm for infants and children. Whether or not the dilatation of the ventricles is the result of increased intraventricular pressure secondary to a decrease in the absorptive area in the subarachnoid spaces, or to a diminution of brain substance secondary to a proliferation of glial tissue with subsequent contracture, has not been definitely determined.

In the study of fixed cerebral lesions it is well to bear in mind that although the encephalogram may give a very definite impression of the existing gross pathologic condition yet it does not give any information as to the etiologic agent responsible for these changes. Trauma, developmental defects, hemorrhage, and infections may all produce pathologic changes which ultimately are similar when studied by this means.

INTRACRANIAL TUMOR

The majority of intracranial tumors occurring in children[89, 90] are located in the cerebellum and usually arise from the roof of the fourth ventricle although occasionally they may arise laterally. Histologically the largest number of tumors may be divided into three groups: (1) Gliomas (medulloblastomas, spongioblastomas, and astrocytomas); (2) Congenital Tumors. The latter are less numerous than the gliomas and arise from an anlage of Rathke's pouch. As a result they seriously interfere with the function of the pituitary body, thereby producing secondary constitutional symptoms. In about 80 per cent of these cases the walls of the cyst become calcified and can be detected by roentgen-ray examination; (3) Tuberculomas. In Cushing's series of 1108 histologically verified tumors only 6 out of 16 tuberculomas occurred in children.

The gliomas are by far the most common tumors occurring during the preadolescent period. Actually they represent 75 per cent of the 154 recorded cases in children in contrast to 37 per cent of 954 cases in adults. Medulloblastomas are considered as arising from embryonic rests, are very malignant, and rapidly metastasize to the meninges producing what is frequently spoken of as "sarcomatosis of the meninges." Spongioblastomas, though equally as malignant, comprise

1 per cent of the tumors in children. The astrocytomas also arise from the roof of the fourth ventricle, are relatively benign, and fortunately are twice as common as the medulloblastomas.

The symptomatology of both of these gliomas shows little variation. Vomiting, usually on arising and before breakfast, is an invariable symptom; headaches are rarely severe or protracted, the patient usually complaining of some suboccipital discomfort or pain on moving the neck; unsteadiness in gait or in the use of the upper extremities is frequently the first symptom to attract attention and may become so severe that the patient is unable to walk without support; papilloedema, which may quickly progress to optic atrophy is present in the majority of early cases and frequently is the only abnormal physical finding. By virtue of their anatomical position these tumors ultimately produce stasis of the cerebrospinal fluid with resulting hydrocephalus.

Prognosis in children with medulloblastomas is hopeless although palliative treatment may give periods of good health. The astrocytomas, if removed completely, yield satisfactory results. An excellent and detailed discussion of intracranial tumors can be found in the cited references.

MENINGITIS

Influenzal Meningitis.—This type of meningitis is looked upon with a great deal of apprehension as extremely few cases recover. Rivers[91] in a review of 220 cases in 1922 found a mortality rate of 92 per cent and subsequent reviews with the inclusion of additional cases have shown only fractional changes in this figure. Bloom[92] in a very extensive review of the literature both in this country and abroad was able to collect twenty-four cases with recovery out of 302 cases. Jenks and Radbill[93] recently reported thirty-two cases treated at the Children's Hospital at Philadelphia over a period from 1921 to 1929 inclusive with no recoveries. Gibbens and Camb[94] in 1931 reported two cases with one recovery.

Ward, Fothergill and Wright in a series of recent papers[95, 96, 97, 98, 99] have made an intensive study of influenzal meningitis from the point of view of producing a therapeutic antiserum, and the study of the mechanism of the action of such a serum. They confirmed the work of early investigators showing the relative homogeneity of influenza bacilli. In addition they have shown that virulent meningeal strains are "smooth" in colony form and produce a specific soluble substance. These latter observations were also made by Pittman.[100]

The above authors have also observed that the presence of agglutinins in the antiserum did not necessarily connote bactericidal activity. Precipitins in the antiserum, however, accurately parallel the bactericidins. In other words, in order to destroy the meningeal strains of influenza bacilli an antiserum must be capable of precipitating the specific soluble substance of virulent strains. The authors succeeded in producing such a serum in horses by prolonged immunization with formalinized virulent influenza bacilli.

In addition they have pointed out that complement is necessary for the bactericidal action of a serum possessing the above-mentioned properties. Complement is practically always absent in the spinal fluid of patients with influenzal meningitis. Their treatment consists

in the administration of antiserum plus fresh normal human serum as complement.

Therapeutic results so far have been encouraging. However, there appears to be early "walling off" in this disease and in many cases, even though it was possible to sterilize the spinal fluid, yet the patients would eventually die from localized lesions. In a personal communication from one of the authors (Fothergill) it has been learned that they have had five complete cures to date. Because of early "walling off" it is needless to emphasize the necessity for early diagnosis.

These investigators have pointed out also certain important statistical data regarding the disease. It is primarily a disease of infancy, about 80 per cent of the cases occurring during the first two years of life. Furthermore, the disease is much more common than is generally supposed, for in their review of meningitis in the Infants' Hospital of Boston they found it the third most common type during the first two years of life. Tuberculous meningitis is the most frequent type with meningococcus meningitis second.

Recently Fothergill and Wright have studied the possible reasons for the striking age incidence of influenzal meningitis, and have found the incidence to be accurately correlated with the bactericidal activity of whole blood from individuals of various age groups. Cord blood and blood during the first six weeks of life possess considerable bactericidal activity for virulent strains. This probably represents a passively acquired humoral antibody transferred from the mother through the placenta and is gradually lost during the first two months of life. It is interesting that the disease practically never occurs during the first two months of life. From two months to three years, the blood has practically no bactericidal activity and it is during this period that the majority of cases occur. From three years on, the killing power gradually rises. This killing power is probably due to an actively acquired immunity represented by a humoral antibody. This antibody, or its absence, is presumably important during the bacteremic stage of the disease.

Streptococcus Meningitis.—Meningitis due to streptococcus infections may be encountered in patients with septicemia but is more frequently seen as a complication of otitis media and mastoiditis.[101] The course is usually rapid and, except in rare instances, is usually fatal. Tawse[102] in 1931, reported the case of an eleven-year-old girl with mastoiditis who recovered. No specific therapy was employed. Rosenberg and Mottley[101] have recently reviewed the literature and carefully analyzed 40 proved cases that recovered. The authors include one patient of their own that was treated with antistreptococcus serum and acriflavine (1:4000 sol.) irrigations of the spinal canal and felt that if the recovery were due to therapy, it probably lay in the use of acriflavine. However, they concluded that since so many different forms of treatment had been used, no particular one was probably of any great value.

Pneumococcus Meningitis.—Pneumococcus meningitis, like streptococcus meningitis continues to have an extremely high mortality rate. Komeda[103] in a study of 40 cases noted that 65 per cent of the cases occurred in infancy, and that 70 per cent of the cases were associated with pneumonia and only 17.5 per cent with otitis media. Recovery occurred in two instances. Davidson and Wollstein[104] in a review of

122 cases treated in the Babies' Hospital at New York City, have made the following observations in regard to the age incidence and group of organism. (Only children under six years of age are admitted.) Fifty per cent of the cases were under six months of age, 24 per cent occurred in the six to twelve months' age group, 23 per cent from one to two years, and 3 per cent in the group over two years of age. Group IV pneumococcus was isolated in twenty-one cases and Group III in one case. In thirty-seven cases the group was undetermined.

Obviously any disease that has as high a mortality rate as pneumococcus meningitis cannot be said to have any specific form of therapy. Mella[105] reports the case of a man who recovered while being treated with four grains of optochin by mouth every five hours accompanied by frequent lavage of the subarachnoid area with Ringer's solution. Weinberg[106] reported success by the use of potassium permanganate retention enemata. (Four ounces of a solution [two grains to twenty-four ounces of water] every four hours.) The author felt that it was undoubtedly of great therapeutic value and strongly urges its use in all such cases. Evans and Welsh,[107] using Huntoon's antibody solution and ethyl hydrocupreine base, unsuccessfully treated two patients. Komer and Amano,[108] as a result of extensive animal experimentation, have made the following recommendations as a prophylactic measure when dealing with severe suppurative foci of the sinuses and middle ear.

"1. A thorough bacteriologic examination should be made of the pus from infected sinuses before operation and of the pus from the mastoid at operation.

"2. In infections of adults with Type I pneumococcus, 10 c.c. of Felton's concentrated antipneumococcus serum should be injected intramuscularly just before or immediately after operation; a second dose should be given the following day and a third dose four days later. Infections with pneumococcus Type II, and Group IV, the same dosage or 15 c.c. should be used; in infections with Type III pneumococcus, a dosage of 20 c.c. should be employed.

"3. The same dosage should be given to children over ten years of age; half doses to younger children.

"4. In infections with pneumococcus, five grains (0.324 gm.) ethyl hydrocupreine hydrochloride with milk should be given every five hours for three days after operation; children require less, according to weight. Although the evidence is not conclusive yet the author feels that the use of ethyl hydrocupreine hydrochloride intravenously and intramuscularly and of ethylhydrocuprine base internally probably enhances the activity of the antipneumococcus serum."

Believing that the intracarotid injection of serum should bring a greater concentration of the material in contact with the organisms, Kolmer[109] treated four patients with pneumococcus meningitis with one recovery. In Type I pneumococcus meningitis he advises the injection of 10 c.c. of Felton's concentrated serum reinforced by 0.5 c.c. of 1:100 optochin hydrochloride into each common carotid artery to be immediately followed by cisternal drainage and injection of 3 to 5 c.c. of the same mixture. If cisternal puncture cannot be made from 10 to 20 c.c. of the mixture should be given intraspinously every eight

hours for the first twenty-four hours. Treatments should be continued for from 2 to 5 days. In the treatment of streptococcus meningitis the same procedure using scarlet fever antitoxin alone is advocated.

Meningococcus Meningitis.—Meningococcus or epidemic meningitis is the one type of purulent meningitis for which we have a specific therapeutic agent; i.e., antimeningococcus serum. However, if complete recovery is to be expected from its use, it must be given at an early date. The classical signs of meningitis are frequently absent in infants and children during that period when serum therapy is so important, and for this reason many patients are not treated until complications have developed. This fact is clearly demonstrated in McKhann's[110] study of 74 cases. In this series there was a mortality rate of 43 per cent, which according to age groups was distributed as follows: Infants under one year of age, 64 per cent; from one to two years, 30 per cent, from two to twelve years, 24 per cent. The significance of these figures is obvious, and their meaning is even more disturbing when one realizes that of the group under two years of age, *48 per cent had been sick over two weeks, and 23 per cent had been sick over 4 weeks before the diagnosis of meningitis was established.* The clinical manifestations of meningeal irritation are usually absent in infants during that period of their illness when serum is most effectual whereas in older children evidence of meningeal irritation appears at an earlier date, and therefore the diagnosis is more quickly established. This variation in the time of appearance of the meningeal manifestations is probably due to the difference in the anatomical structure of the head of an infant and that of a child. In the former the sutures are not ossified and the fontanel is open, while in the latter these conditions do not exist. As a result of the expansile ability of the cranial vault of the infant increased intracranial pressure manifests itself at a later date than in the child whose cranial vault is rigid and nonexpansile. Therefore, the earliest evidence of the disease in an infant is that of infection, whereas in the child it is that of increased intracranial pressure. The present high mortality rate of this disease should be reduced if one considers this difference, and considers the possibility of meningitis in infants whose illness is not otherwise satisfactorily explained.

The treatment of this type of meningitis varies according to whether one is dealing with an epidemic or with the usual sporadic case. In times of an epidemic the organism is more virulent and therefore more drastic measures may be necessary. Ordinarily treatment by the spinal subarachnoid route either with or without ventricular or cisternal injections[110] will prove adequate. During the recent epidemic 1067 patients were treated in the Herman Kiefer Hospital at Detroit, and many cases were encountered that did not respond to the usual form of treatment but did respond when the serum was given by the intraspinal intracisternal and intravenous routes. Gordon,[111] in his discussion of the results by serum treatment of this large series states, "Several routes have been described for administration of antimeningococcus serum. Opinion varies as to the methods productive of best results. Some recommend a conservative method employing daily injections of the serum intraspinously. Others feel, because of the septicemic phase of the disease, that with early diagnosis, serum should be administered both intravenously and intraspinously. Because of mechanical difficulties in

the circulation of the spinal fluid in infants a method of combined intraventricular and intraspinous injection has been advocated for meningitis in that age group.

"No single definite program is applicable to all conditions. As with most infectious processes, treatment is best guided by the individual clinical manifestation of the particular patient and the general severity of the existing epidemic. In ordinary circumstances the most satisfactory results with any disease are obtained by conservative measures. Under unusual circumstances there should be no hesitation in using more energetic forms of treatment."

In the serum therapy of meningococcus meningitis, it is extremely important that the serum employed agglutinates the organisms of the strain causing the infection. This can be determined in a relatively short period of time by laboratory procedures. If the serum does not agglutinate the organism one should obtain a serum that possesses this quality, otherwise specific therapy will be of little value. Many failures in the past can probably be explained on this basis.

TETANY

In considering the conditions that may precipitate convulsions during the first few days or weeks of life one does not ordinarily think of tetany, as this disturbance is usually associated with rickets. However recent case reports have shown that a clinical syndrome simulating infantile tetany may occur not only during the first few weeks of life, but even within the first few days.

Bass and Karelitz[112] recently reported three newborn infants all of who presented the classical clinical picture of tetany, i.e., convulsions, carpopedal spasms, Chvostek's[113] sign and laryngeal crow. In these infants vomiting was such a prominent symptom that intestinal obstruction had to be excluded, and the authors suggested that the condition might have been secondary to the loss of HCl and fixed base in the vomitus, with resulting decrease in the serum chloride concentration, increase in bicarbonate, and decrease in fixed base of the blood.[114] McGavran[115] has recently reported the case of an infant that presented the clinical manifestations of tetany 36 hours after birth. Unfortunately serum calcium determinations were not included in these case reports. However, in each case there was an immediate and dramatic response to the therapeutic test; namely, the administration of calcium chloride. Powers[116] has reported a five-week-old infant that presented this syndrome and who had a blood serum calcium of 5.3 mg. per 100 c.c. of blood. Small[117] has observed a nine-day-old infant who presented the typical clinical manifestations of tetany and who had a blood serum calcium of 5.60 mg. per 100 c.c. The administration of calcium chloride promptly relieved the symptoms.

From the reports it is obvious that one must not only consider intracranial hemorrhage as a cause of convulsions in newborn infants but also tetany. Should the clinical manifestations suggest the latter, proper therapy should be instituted.

REFERENCES

1. Sherwood, David W.: Chronic Subdural Hematoma in Infants, Am. J. Dis. Child. **39**: 980-1021, 1930.
2. Hunt, F. C.: Internal Haemorrhagic Pachymeningitis in Children, Am. J. Dis. Child. **39**: 84-90, 1930.

3. Peet, Max M., and Kahn, Edgar H.: Subdural Hematoma in Infants, J. A. M. A. **98**: 1851-1856, 1932.
4. McKhann, Charles F.: Meningococcus Meningitis in Infants, New England J. M. **202**: 520-523, 1930.
5. Bagley, Charles, Jr.: Blood in Cerebrospinal Fluid: Resultant Functional and Organic Alterations in the Central Nervous System, Arch. Surg. **17**: 18, 1928.
6. Bagley, Charles, Jr.: Spontaneous Cerebral Hemorrhage: Discussion of Four Types with Surgical Considerations, Arch. Neurol. and Psychiat. **27**: 1133, 1932.
7. Rosenberg, O.: Ergebn. d. inn. Med. u. Kinderh. **20**: 549, 1921.
8. Burhans, C. W., and Gerstenberger, H. J.: Internal Haemorrhagic Pachymeningitis in Infancy, J. A. M. A. **80**: 604, 1923.
9. Crothers, Bronson: Personal Communication.
10. Chase, W. H.: An Anatomical Study of Subdural Haemorrhage Associated with Tentorial Splitting in the Newborn, Surg., Gynec. and Obst. **51**: 31, 1930.
11. Goldzieher, M. A., and Gordon, M. B.: Syndrome of Adrenal Hemorrhage in the Newborn, Endocrinology **16**: 165, 1932.
12. Clifford, Stewart H., and Hertig, Arthur T.: Erythroblastosis of the Newborn, New England J. M. **207**: 105-113, 1932.
13. Munro, Donald: Cranial and Intracranial Damage in the Newborn, an End Result Study of 117 Cases, Surg., Gynec. and Obst. **47**: 622, 1928.
14. Grulee, Clifford: The Diagnosis of Intracranial Haemorrhage in the Newborn, New York State J. Med. **31**: 1032-1036, 1932.
15. Fleming, G. B.: Birth Injuries of the Newly Born, Lancet **2**: 532, 1931.
16. Gillespie, James B.: Stillbirths. Analysis of the Causes of Death in 338 Cases as Determined by Necropsy, Am. J. Dis. Child. **44**: 9-15, 1932.
17. Ehrenfest, Hugo: The Relation of Birth Trauma to Neonatal Mortality and Infant Morbidity, Am. Jour. Dis. Child. **43**: 426-463, 1932.
18. Irving, Frederick C.: The Obstetrical Aspect of Intracranial Hemorrhage, New England Jour. Med. **203**: 499-502, 1930.
19. Eden, Thomas W.: The Infant Mortality of Birth, Lancet **2**: 1223-1227, 1931.
20. Clifford, Stewart H.: Personal Communication.
21. Yagi, Hideo: Birth Injuries in the Newborn (series), Jap. J. Obst. & Gynec., 1926-1931.
22. Crothers, Bronson: Mechanism of Labor from the Neurologic Point of View, J. A. M. A. **92**: 99, 1929.
23. Ford, Frank R.: Cerebral Birth Injuries, Medicine Monograph Vol. XI, Baltimore, Williams & Wilkins, 1927.
24. Schroeder, P. L.: Behavior Difficulties in Children Associated with the Results of Birth Trauma, J. A. M. A. **92**: 100, 1929.
25. Winkelman, N. W.: Cerebral Trauma and Its Relation to Cerebral Deficiency, Am. J. Psychiat. **10**: 611-621, 1931.
26. Crothers, Bronson, et al.: Encephalography in Cases with Fixed Lesions of the Brain, Am. J. Dis. Child. **40**: 227-246, 1930.
27. Crothers, Bronson, and Putnam, Marian C.: Cord Birth Injuries, Medicine **6**: 4, 1927.
28. Byers, Randolph K.: Late Effects of Obstetrical Injuries at Various Levels of the Nervous System, New England J. Med. **203**: 507-522, 1930. Ibid Transection of the Spinal Cord in the Newborn, Arch. Neurol. and Psychiat. **27**: 585-592, 1932.
29. Husler, J., and Spatz, H.: Ztschr. f. Kinderh. **38**: 428, 1924.
30. Jochims, J.: Ztschr. f. Kinderh. **45**: 326, 1928.
31. Ford, F. R.: Am. J. Dis. Child. **37**: 1046, 1928.
32. Askin, J. A., and Zimmerman, H. M.: Am. J. Dis. Child. **38**: 97, 1929.
33. Singer, Ludwig: Ztschr. f. Kreislaufforsch. **20**: 338, 1928.
34. Cabot, Richard C., and H.: Boston Med. and Surg. J. **194**: 634, 1926.
35. Blühdorn, K.: Ztschr. f. Kinderh. **26**: 251, 1920.
36. Powers, G. F.: Am. J. Dis. Child. **30**: 632, 1925.
37. Eley, R. Cannon: New England J. M. **203**: 162-167, 1930.
38. Gordon, M. H.: Experimental Production of the Meningoencephalitis of Mumps, Lancet **1**: 652, 653, 1927.
39. Haden, Russell L.: The Cerebral Complications of Mumps, Arch. Int. Med. **23**: 737-744, 1919.

40. Casparis, Horton R.: Cerebral Complications in Mumps, Am. J. Dis. Child. **18:** 187-193, 1919.
41. Miller, Milo K.: Four Types of Encephalitis, J. A. M. A. **97:** 161, 1931.
42. Rolleston, J. D.: Some Aspects of Mumps: A Review of the Recent Literature. Practitioner **128:** 31-39, 1932.
43. Zimmerman, H. M., and Yannet, Herman: Nonsuppurative Encephalomyelitis Accompanying Chicken-Pox, Arch. Neurol. and Psychiat. **26:** 322-332, 1931.
44. Ford, F. R.: The Nervous Complications of Measles, Bull. Johns Hopkins Hosp. **43:** 140, 1928.
45. Heath, Parker: Measles Encephalitis: A Clinical Report of Some Eye Findings, Am. J. Ophth. **15:** 130-134, 1932.
46. Greenfield, J. G.: Brain **52:** 171, 1929.
47. Ferraro, A., and Scheffer, I. H.: Arch. Neurol. and Psychiat. **25:** 748-778, 1931. Ibid. Arch. Neurol. and Psychiat. **27:** 1209-1225, 1932.
48. Troup, A. G., and Hurst, E. W.: Disseminated Encephalomyelitis Following Smallpox, Lancet **1:** 566, 1930.
49. McIntosh, J., and Searff, R. W.: The Reaction of the Central Nervous System to Vaccinia Virus, J. Path. & Bact. **33:** 483-488, 1930.
50. Eckstein, A.: Investigations on the Causes of Vaccination Encephalitis, Arch. Dis. Childhood **7:** 105-116, 1932.
51. Jorge, Recordo: Postvaccinal Encephalitis, Lancet **2:** 215-219, 1932. Ibid. Lancet **2:** 267-270, 1932.
52. Flexner, Simon: Postvaccinal Encephalitis and Allied Conditions, J. A. M. A. **94:** 305-311, 1930.
53. Thompson, Richard: The Etiology of Postvaccinal Encephalomyelitis, Arch. Path. **12:** 601-632, 1931.
54. Miller, M. K.: Four Types of Encephalitis, J. A. M. A. **97:** 161, 1931.
55. Gordon, A. H.: Postvaccinal Encephalitis, Tr. A. Am. Physicians **46:** 188-196, 1931.
56. McKhann, Charles F.: Lead Poisoning in Children; The Cerebral Manifestations, Arch. Neurol. and Psychiat. **27:** 294-303, 1932.
57. Ruddock, J. C.: Lead Poisoning in Children with Special Reference to Pica, J. A. M. A. **82:** 1682, 1924.
58. Wilcox, H. B., and Caffey, J. P.: Lead Poisoning in Nursing Infants; Report of Two Cases Due to Lead Nipple Shields, J. A. M. A. **86:** 1514, 1926.
59. Weller, C. V.: Tolerance in Respect to Meningocerebral Manifestations of Acute and Subacute Lead Poisoning: Experimental Production, Arch. Int. Med. **39:** 45, 1927.
60. Kehoe, R. A., and Thamann, F.: The Excretion of Lead, J. A. M. A. **92:** 1418, 1929.
61. Park, Edwards A., Jackson, Deborah, and Kajdi, Laslo: Shadows Produced by Lead in the X-ray Pictures of the Growing Skeleton, Am. J. Dis. Child. **41:** 485-499, 1931.
62. Vogt, Edward C.: A Roentgen Sign of Plumbism. Am. J. Roentgenol. **24:** 550-553, 1930. Ibid. Roentgenologic Diagnosis of Lead Poisoning in Infants and Children, J. A. M. A. **98:** 125-128, 1932.
63. Caffey, John: Clinical and Experimental Lead Poisoning: Some Roentgenologic and Anatomic Changes in Growing Bones, Radiology **17:** 957-983, 1931.
64. Aub, J. C., Fairhall, L. T., Minot, A. S., and Reznikoff, P.: Lead Poisoning, Medicine **4:** 1, 1925.
65. Phemister, D. B.: The Effect of Phosphorus on Growing, Normal, and Diseased Bones, J. A. M. A. **70:** 1737, June 18, 1918.
66. Hunter, D., and Aub, J. C.: Lead Studies XV. The Effect of the Parathyroid Hormone on the Excretion of Lead and of Calcium in Patients Suffering from Lead Poisoning, Quart. J. Med. **20:** 123, 1927.
67. McKhann, Charles F.: Lead Poisoning in Children. With Notes on Therapy, Am. J. Dis. Child. **32:** 386-392, 1926.
68. Gamble, J. L., Ross, S. G., and Tisdall, F. F.: Studies of Tetany, Am. J. Dis. Child. **25:** 455, 1923.
69. Cobb, S.: Causes of Epilepsy, Arch. Neurol. and Psychiat. **27:** 1245, 1932.
70. Fay, Temple: Some Factors in the "Mechanical Theory of Epilepsy" with Especial Reference to the Influence of Fluid and Its Control, in the Treatment of Certain Cases, Am. J. Psychiat. **8:** 783, 1929.
71. McQuarrie, Irvine: Epilepsy in Children: The Relationship of the H_2O Balance to the Occurrence of Seizures, Am. J. Dis. Child. **38:** 451, 1929.

McQuarrie, Irvine, Manchester, R. C., and Husted, Clara: The Effect of Induced Changes in the State of Hydration Upon the Occurrence of Convulsions in Epileptic Children, in Epilepsy, and the Convulsive State, Proc. A. Research Nerv. and Ment. Dis., Baltimore, Williams & Wilkins Co., 1932, Vol. 7, p. 623.

72. Fremont-Smith, Frank.: Epilepsy and the Convulsive State. Baltimore, Williams & Wilkins Co., p. 617, 1931.
73. McQuarrie, Irvine, Manchester, R. C., and Husted, Clara: Study of the Water and Mineral Balances in Epileptic Children, Am. J. Dis. Child. **43**: 1519, 1932.
74. Fay, Temple: Epilepsy: Clinical Observations on the Control of the Convulsive Seizures by Means of Dehydration, Jour. Nerv. and Ment. Dis. **71**: 481-627, 1930.

Therapeutic Effects of Dehydration on Epileptic Patients, Proc. A. Research Nerv. and Ment. Dis. Baltimore, Williams & Wilkins Co., 1932.

75. Peterman, M. G.: Ketogenic Diet in the Treatment of Epilepsy, Am. J. Dis. Child. **28**: 28-33, 1924.
76. Helmholz, H. F.: J. A. M. A. **88**: 2028, 1927.
77. Talbot, F. B., Metcalf, K. M., and Moriarty, M. E.: Boston M. & S. J. **196**: 89, 1927.
78. Peterman, M. G.: Epilepsy in Children, Wisconsin M. J. **31**: 169, 1932.
79. McClintic, Clifton G.: A Surgical Treatment of Epilepsy, Proc. A. Research Nerv. and Ment. Dis., Baltimore, Williams & Wilkins Co., p. 659, 1932.
80. Foerster, O., and Penfield, Wilder: The Structural Basis of Traumatic Epilepsy and Results of Radical Operation, Proc. A. Research Nerv. and Ment. Dis., Baltimore, Williams & Wilkins Co., p. 569, 1932.
81. Dandy, W. E.: Ventriculography Following the Injection of Air Into the Cerebral Ventricles, Ann. Surg. **68**: 5, 1918.
82. Sharp, E. A.: Artificial Pneumorachis in the Treatment of Acute Infections of the Meninges, Arch. Neurol. and Psychiat. **6**: 669, 1921.
83. Penfield, W.: Chronic Meningeal (Posttraumatic) Headache and Its Specific Treatment by Lumbar Air Insufflation, Surg., Gynec. and Obst. **45**: 747, 1927.
84. Lyon, George M.: Probable Aqueduct Obstruction Occurring in Meningococcus Meningitis, J. A. M. A. **95**: 109, 1930.
85. Bogin, Maxwell, Holzsager, Theodore, and Kramer, Benjamin: Encephalography in Children, Am. J. Dis. Child. **42**: 526, 1931.
86. Crothers, Bronson, Vogt, Edward, and Eley, R. Cannon: Encephalography in Cases with Fixed Lesions of the Brain, Am. J. Dis. Child. **40**: 227-246, 1930.
87. Eley, R. Cannon, and Vogt, Edward: Encephalography in Children; Further Observations in Children with Fixed Lesions of the Brain, Am. J. Roentgenol. **27**: 686-696, 1932.
88. Pancoast, H. K., and Fay, T.: Encephalography: Roentgenological and Clinical Considerations for Its Use, Am. J. Roentgenol. **21**: 421, 1929.
89. Bailey, Percival, and Cushing, Harvey: Medulloblastoma Cerebelli, Arch. Neurol. and Psychiat. **14**: 192-223, 1925.
90. Cushing, Harvey: The Intracranial Tumors of Preadolescence, Am. J. Dis. Child. **33**: 551-584, 1927. Experiences with Cerebellar Medulloblastomas, Acta Pathologica et Microbiologica Scandinavia, Vol. 7, 1930.

— Experiences with the Cerebellar Astrocytomas, Surg., Gynec. and Obst., **52**: 129-204, 1931.

— Intracranial Tumors, Charles C. Thomas, Springfield, Ill., 1932.

91. Rivers, T. M.: Influenzal Meningitis, Am. J. Dis. Child. **24**: 102, 1922.
92. Bloom, C. J.: Influenzal Meningitis Is Amenable to Treatment, New Orleans M. and S. J. **83**: 455-467, 1931.
93. Jenks, H. H., and Radbill, S. X.: Influenzal Meningitis, Arch. Pediat. **48**: 1-15, 1931.
94. Gibbens, J.: Influenzal Meningitis, Lancet **1**: 291-293, 1931.
95. Ward, H. K., and Fothergill, L. D.: Influenzal Meningitis Treated with Specific Antiserum and Complement, Am. J. Dis. Child. **43**: 873-881, 1932.
96. Ward, H. K., and Wright, Joyce: Studies on Influenzal Meningitis. I. The Problems of Specific Therapy, J. Exper. Med. **55**: 223-234, 1932.
97. Fothergill, L. D., Wright, J., and Ward, H. K.: The Specific Treatment of Influenzal Meningitis, Abs. J. Bact. **23**: 99, 1932.

98. Wright, J., and Ward, H. K.: Studies on Influenzal Meningitis, II. B. Influenza. The Problem of Virulence and Resistance, J. Exper. Med. **55**: 235-246, 1932.
99. Fothergill, L. D., and Wright, J.: Influenzal Meningitis. The Relation of the Incidence of the Disease to the Bactericidal Power of the Blood for the Causal Organism. (In press).
100. Pittman, M.: Variation and Type Specificity in the Bacterial Species Hemophilus Influenzae, J. Exper. Med. **53**: 471-491, 1931.
101. Rosenberg, L., and Nottley, H. W.: Recovery from Streptococcus Meningitis, Ann. Int. Med. **4**: 1154-1182, 1931.
102. Tawse, H. Bell: Streptococcus Otitic Meningitis, J. Laryng. & Otol. **46**: 481, 482, 1931.
103. Komeda, M.: The Clinical Study of Pneumococcus Meningitis in Children, Oriental J. Dis. Infants **9**: 31-33, 1931.
104. Davidson, L. T., and Wollstein, Martha: Pneumococcus Meningitis in Children with an Analysis of 122 Cases, Acta Pediat. **11**: 367-369, 1930.
105. Mella, Hugo: The Use of Subarachnoid Lavage and Ethylhydrocuprine in Meningitis, U. S. Vet. Bur. M. Bull. **7**: 77, 1931.
106. Weinberg, Max H.: A Case of Pneumococcus (Type III) Meningitis Treated with Potassium Permanganate—Recovery, J. Nerv. and Ment. Dis. **74**: 38-45, 1931.
107. Evans, J. A., and Welsh, S. N.: Treatment of Septic Meningitis by Intracarotid Serum Therapy, Ann. Int. Med. **4**: 1308-1312, 1931.
108. Kolmer, John A., and Amano, K. W.: The Specific Prophylaxis of Pneumococcus and Streptococcus Meningitis, Arch. Otolaryng. **14**: 125-138, 1931.
109. Kolmer, John A.: The Intracarotid Method of Treatment of Meningitis with Recoveries, J. A. M. A. **96**: 1358-1361, 1931.
110. McKhann, Charles F.: Meningococcus Meningitis in Infants, New England J. Med. **202**: 520-523, 1930.
111. Gordon, J. E.: Herman Kiefer Hospital—Detroit Department of Health—Bureau of Epidemiology. Monthly Report for November, 1931.
112. Bass, Marray H., and Karelitz, Samuel: Tetany Accompanied by Hyperpyrexia and Vomiting in the First Few Days of Life, J. A. M. A. **97**: 1372-1375, 1931.
113. Stevenson, Frank E., Mitchell, A. Graeme, and Koch, Carl A.: Chvostek's Sign in Newborn Infants, Am. J. Dis. Child. **34**: 425-428, 1927.
114. Gamble, James L., and McIver, Monroe A.: A Study of the Effects of Pyloric Obstruction in Rabbits, J. Clin. Investigation **1**: 531, 1925.
115. McGraven, E. G.: Spasmophilic Tetany in the Newborn, J. A. M. A. **99**: 115, 116, 1932.
116. Powers, G. F.: Tetany as a Cause of Convulsions in Very Young Infants, J. A. M. A. **84**: 1907, 1925.
117. Small, Abraham: Personal Communication. Case to be reported.

American Academy of Pediatrics

Proceedings

SECOND ANNUAL MEETING OF THE AMERICAN ACADEMY OF PEDIATRICS

FRIDAY AFTERNOON SESSION

May 13, 1932

Round Table Conference on Disorders of the Blood and the Hemopoietic System

Leader: Dr. Thomas B. Cooley; Assistants: Wm. M. Happ and Heyworth N. Sanford.

CHAIRMAN COOLEY.—We cannot, of course, in a single afternoon, discuss all the disorders of the hemopoietic system. It seems to me that we can profitably give most of our time to that part of our subject which has the greatest general interest—the anemias of infancy and early childhood—taking up later such other special subjects as time permits.

Approaching a discussion of the anemias, I think we should try so far as possible to look at them from an etiologic viewpoint. For a long time we have talked of anemias as "primary" and "secondary." As we have learned more about them we have found that some of those we once called primary are really secondary to conditions outside the hemopoietic system, and some of the "secondary" disorders are possibly due to fundamental faults of the blood-forming mechanism itself. That particular classification is hardly worth keeping. Most of our names for the anemias are not derived from their etiology, and in that sense are not very satisfactory, but many of them, through long use, have come to connote pretty definite clinical entities, and so are likely to stick. Others, applied to conditions loosely if at all related in etiology, will be replaced as our knowledge grows.

I have jotted down a simple classification of the etiologic factors in the production of anemia. It is not my own—in fact, I am indebted to Isaacs for most of it—and I should not call it perfect, but it will give us a basis for our consideration.

First, of course, must come the most obvious and typical of all causes for anemia—direct blood loss. This may be acute or chronic; hemorrhage from trauma, bleeding over a short period in purpura, the hemorrhagic disease of the newborn, etc.; or the more prolonged loss in enteritis and other ulcerative processes. This, the most typical, is also the best understood of the anemias, and therefore the least important to the present discussion.

Second, I should put blood destruction—by toxins, gaseous, mineral, drug and bacterial—due also to congenital or acquired perversions of the normal process of blood destruction. These are the "hemolytic" anemias.

Third, a failure of normal blood production, giving rise to the so-called "aplastic" anemias. Aplastic anemia, as the name is commonly used, denotes a

very grave condition; but there are all sorts of lesser grades of aplasia, in which the marrow activity has been temporarily interfered with. These have been included in the loose group of "secondary" anemias, but it is desirable to distinguish them when this is possible.

Fourth, comes a classification in which I do not myself believe, but which is in common use; the "nutritional" anemias. There have been various ideas as to the effect of diet in causing anemia, such as Glanzmann's theory of a hemolytic action of the unsaturated fatty acids of cow's milk, and some of the bizarre explanations advanced for "goat's milk anemia." Dr. Sanford is to talk about nutritional anemia, and I don't know what he will say, but I believe that practically all of the conditions that have been classed here belong in what I have come to think of as another group—the deficiency anemias.

Fifth, this group of deficiency anemias comprises those in which the anemia is due to a defect in the supply to the marrow of the materials requisite for the production of enough complete cells. The deficiency may lie in defective diet, poor absorption, or some failure in intermediate metabolism. Present knowledge of pernicious anemia puts it quite obviously in this group. Erythroblastic anemia may belong here. Analysis of the various types of "nutritional anemia" shows quite clearly, I think, that they are nearly all due to a simple and obvious deficiency—a lack of iron.

The *Sixth* cause of anemia is a crowding out of the hemopoietic marrow by infiltration of the marrow cavity with pathologic growths, leucemic tumors, the metastases of malignancy, etc. These are the myelophthisic anemias.

While these are the main etiologic factors in the anemias, and I think it desirable to think of the clinical manifestations from this viewpoint, it is by no means always possible to put a particular case into a single etiologic group, for combinations of causes are rather common. The anemias with which we most frequently have to deal, those developing in the course of infection, are probably usually due to blood destruction (hemolysis) plus diminished production (aplasia). During the infection these two are at work together; in the postinfection period, though excessive hemolysis may no longer be present, the aplasia due to the action of toxins of infection on the marrow may persist for a long time, and the resulting anemia be slow to disappear. Josephs had a very interesting paper last year showing the dependence of these common anemias on this lack of balance between production and destruction.

There is another thing that is being said rather commonly nowadays. That is, that the only way in which we can satisfactorily account for severe anemia developing sometimes in childhood from apparently trivial causes is to suppose what one might call an anemic diathesis—a peculiar propensity of certain children to develop a particular type of anemia—aplastic or hemolytic. For some reason the reticuloendothelial system of these children is peculiarly liable to be upset in some certain way. This seems to be the most satisfactory explanation of many clinical observations.

So much for the things that can be classified. We have still a group of what my friend Dr. Janet Vaughan calls "wastebasket diagnoses," and that's a good term. There are whole groups of anemias about which we still do not know enough to classify them definitely from any etiologic viewpoint, so that we still make free use of these wastebasket diagnoses, and I don't know want we should do without them. One of the best is "splenic anemia." It covers a multitude of sins. I am thinking of the anemia with splenomegaly and leucopenia which occurs in young children and resembles closely the description of the first stage of Banti's disease. One sees them rather often, and usually doesn't know the reason for them. There is more than one kind of splenic anemia. I believe that Strümpell at various times

described three, and Banti himself two, so that one can hardly use the term as signifying any definite entity; but it is still in use all over the world for these little understood cases in childhood.

A thing that has been attracting attention recently, which seems still to belong in this class of wastebasket diagnoses is congenital anemia. This is a severe anemia developing soon after birth. A number of reported cases has accumulated, and Dr. Happ is to tell us something about them, but I doubt if he will tell us their exact cause.

Another condition attracting present interest is the erythroblastosis of the newborn—a disease characterized by a tremendous number of nucleated red cells and a very high degree of hemolysis. I want to say a few words about this later. We have no present basis for classifying it etiologically.

Some other disorders of the hemopoietic system do not belong strictly in classifications of the anemias; that is, their principal symptoms arise not from lack of red cells and hemoglobin, but from disturbances of one sort or another in the white cells. Some of these white cell disorders parallel very closely those of the red cells. The likeness between thrombolytic purpura and hemolytic icterus, for instance, is striking.

I had hoped that Dr. Buckman would be here. He was down, as you know, as one of the assistants in this program, and has been studying the physiology of the blood in early infancy. This is a subject I should like to have discussed, and I do not feel competent to do it myself. I can only say a word to introduce the subject.

Obviously, in considering blood disturbances in very early life, one has to think of the changes at the time of birth, and their possible relation to the anemias of infancy. The blood in intrauterine life does not carry on its important function, that of the respiratory interchange, in the same manner as after birth. At the time of birth it has to adjust itself to a very sudden change, and this adjustment may not always be perfect. That, to my mind, is a logical explanation of some of the things we see; that, and another thing that may occur; namely, that the hemopoietic system may not have reached its full normal development at birth, and may be inadequate to its new duties. I think we must consider the anemias occurring soon after birth in relation to these questions, and perhaps in relation to some disorders of the maternal organism, concerning all of which we know very little.

Before we go on to talk of specific anemias, I should like to put in a plea for more exactness in the common language of hematology. It may be too much to ask that we should all use the same disease classification at present, or the same cytologic nomenclature, but I see no reason why we should not use precise terms for one of our most important observations—the amount of hemoglobin. It is still almost universal to express this in terms of percentage, which might be all right if everyone employed the same method and the same standard. Unfortunately, this is not the case. There are two standards in common use, and two scales. The situation is like that of the systems of weights and measures in use in different countries, except that often, in reading case reports of anemias, we have no indication as to what system the author used, and are consequently hampered in making comparisons. It is simple enough to report hemoglobin in grams per 100 c.c., and I hope this method may soon come into general use.

We come now to consideration of some diseases of special interest, and will take up first "congenital anemia." Dr. Happ has had some experience with this, and has had occasion to familiarize himself with the literature.

DR. HAPP.—Dr. Cooley has mentioned loss of blood and blood destruction as two of the chief factors in the production of anemia. I think in the cases under

discussion we are not dealing with either of these factors. There is no evidence of loss of blood, but rather of defective blood formation.

Certain infants, born of apparently normal mothers, after a more or less normal delivery, develop anemia. They appear to be healthy babies at birth, have good color and are not jaundiced. Blood counts are not done routinely on normal newborn babies, so there is no actual proof that these babies do not have anemia when they are born. But at least they do not have sufficient pallor to attract the attention of the doctor or nurse. At about the fifth or the seventh day the nurse or interne or attending physician notices that the child is quite pale and subsequent blood counts show an extreme degree of secondary anemia, without any particular change in the white cells. In other words, the white cell formation goes on at a normal rate, but the red cells are greatly reduced in number and in hemoglobin content. The spleen is usually not enlarged at this stage, that is, between the fifth and the tenth days of life. If these babies are transfused, according to available reports, recovery generally ensues, but if they are not transfused they are apt to succumb to a diminution of red cells to a point that is not compatible with life. There is no jaundice and no evidence of hemolysis.

Dr. Arthur Abt collected sixteen of these cases which he reported in the *American Journal of Diseases of Children* a few months ago. Of those sixteen cases, only two died, and the remaining fourteen recovered. No information was given in this collection as to why these babies did not manufacture blood cells in the normal manner. Recently I had the opportunity to study the pathologic findings in a case of this type. This was not the case which I reported that was included in Abt's series, but another baby, who was born of a mother thirty-five years old who had previously given birth to a baby who died on the seventh day of a similar condition. So it is possible that a mother may give birth to two children showing the same peculiar lack of blood formation. The first baby had died at the age of seven days. This baby was born at term and pallor was noted at about the fifth day. A small amount of blood was given intramuscularly but the baby was not transfused. Death occurred on the tenth day. I did not see the patient during life. At autopsy, the spleen was found somewhat enlarged, weighing thirty grams. No enlargement of the lymph glands was noted. The bone marrow was pale and sections showed marked deficiency in erythropoiesis. There were few red cells in the marrow and few megakaryocytes, megaloblasts and red cell precursors. The liver showed a great deal of erythroblastic activity, islands of cells scattered throughout the liver apparently in a futile attempt to manufacture red cells in a fetal manner. This condition was noted also in the spleen.

From this case and from an analysis of the reported cases, there is apparently a congenital deficiency of red cell forming elements in the bone marrow of these babies, so that the red cells which are manufactured are immature and short-lived. After birth the marrow does not make more red cells in the normal manner so that the cells that are present at birth gradually deteriorate and an increasing anemia develops. With the stimulation by transfusion, the infant manufactures red cells and gets along satisfactorily. The cases which recovered remained well throughout infancy.

That is about the status of these cases. As to their classification, I do not feel that they constitute a definite entity. I feel that anemias in newborn babies comprise a heterogeneous group, which may have a variety of etiologic factors. There may be factories in the maternal organism which are responsible in some cases. I feel that it is possible to have babies with this picture in a modified degree which does not express itself during the lying-in period, but which may show itself later. Some of the nutritional anemias which develop during the first year may have this etiology in a modified manner, so that erythropoiesis may occur subnormally. I

think it is wrong to regard this as a specific group or specific disease, but simply as a collection of heterogeneous groups of anemias which are similar but which are not necessarily identical.

DR. SANFORD.—I have reported three of these cases. The first which I ran into was reported in 1925. That case was picked up accidentally. I thought the child was normal, but Dr. Grulee said the child was anemic. We took the blood count and it proved to be so. The hemoglobin was 50. We followed the case and took a blood count about three years later. The hemoglobin was 75 per cent and the red cells were markedly increased. In the original findings there were no other symptoms than marked anemia.

We kept the first child in the hospital about a month. The other two were discharged in fifteen or twenty days with greatly increased hemoglobin and red cells. The first patient had no treatment at all, except that we made röntgenograms of the long bones. Whether momentary exposure to the x-ray could have had any effect, I could not say. I followed this child for six years. The last blood count last fall was perfectly normal. As to the other two children; we were doing at that time routine ultraviolet exposure to see whether it would have any effect on the hemoglobin and the red cells. They did, for a few days, receive short exposures. The anemia apparently cleared itself without any difficulty. I have not been able to follow those two latter cases.

DR. GRULEE.—I saw one case diagnosed at one hour of age. It was very evidently a congenital anemia. I have seen three types of anemia in the newborn clinical types, you might say. One is the type which Dr. Sanford just discussed and in which this last case I mentioned comes. I saw one child, previously apparently a normal baby, which on the ninth day developed a very severe anemia and died on the tenth day. It was of the so-called aplastic type of anemia. Complete autopsy failed to reveal anything but anemic organs. The other type of anemia which has been particularly bad and of which I have seen two cases is the so-called erythroblastosis.

DR. McGEE (RICHMOND).—I have seen one so-called congenital anemia which was brought in from a country district. The child was five weeks old. It was panting for breath. I immediately thought of congenital heart disease but found no signs. The child did not get blue but was always pale. Sometimes as often as every half hour the infant would breathe rapidly. The breathing was like that seen in a respiratory disease. The red cell count at five weeks was 2,900,000, the hemoglobin 52 per cent (Sahli), white cells, 10,400, and there were a few normoblasts, megaloblasts and myelocytes. The shift to the left was 45 per cent. The child was given copper and iron, which he could not hold. The dose was cut and still he could not retain it. He was then given liver extract and ultraviolet light. Apparently the latter two measures helped. He was on the bottle entirely when I saw him and had not been breast-fed for a couple of weeks. The red cell count went to 5,120,000 cells, while the hemoglobin increased from 52 to 70 per cent. With the increase in the number of red cells and in the hemoglobin there was a subsequent decrease in the young white cells and in the shift to the left.

At six months of age the child had stopped panting and looked very well. It had been very pale. The physician who delivered the child said it would probably outgrow the panting. I did not know what the trouble was at first but felt the anemia would probably account for the panting as the lungs were trying to get more air.

DR. HILL.—Do you give liver extract by mouth?

DR. McGEE.—Yes, I gave it by mouth. I used Neobovinine. I happened to have some on hand and some of the physicians in Richmond said they thought it

was good in spite of the fact that it was not recognized by the Council of the A. M. A. I gave about fifteen drops three times a day.

DR. HAPP.—I do not think that the anemias of this type are nearly so rare as one would gather from the number of reported cases.

DR. COOLEY.—I think the reason why we do not see them in our clinic is that we have no connection with a lying-in hospital and see very few newborn babies. I think the anemia might just as well occur in prematures.

DR. HAPP.—Only one of the sixteen, Dr. Foote's case, was premature.

DR. GRULEE.—I think I can give you some idea on the rarity of it. We have about thirty obstetrical cases a month, and for several years we did a complete blood count on every newborn. We didn't have a case until that first reported one, so that it is not common; but we have had three or four more cases. When you are dealing with the newborn, you are dealing with a number of normal patients. It is not as if you were picking one or two from an abnormal group.

DR. COOLEY.—I want to say a word or two about a thing that Dr. Grulee mentioned. That is this so-called erythroblastosis of the newborn. It is a condition of which we have heard little until lately, but apparently it is not so uncommon. I know that men who have been interested in it in Boston have seen quite a few cases. I have had the interesting experience of seeing two in a few weeks. The reason I wanted to speak about it is that we have had what I think is a unique experience with one of them. That is, we cured it, temporarily, by splenectomy. We did cure the anemia. The child died of something else.

This condition is a striking one, which I think has often been missed because it has been allowed to go with a diagnosis of icterus gravis. That diagnosis has been made and the condition not carefully studied. In both of the cases which I saw lately other children in the families had died of what seems to have been the same thing. The histories were quite typical of familial icterus gravis. The children became jaundiced within a few hours after birth. The color increased very rapidly in depth. There were enormous numbers of nucleated red cells and a very high degree of hemolysis, the icterus index having gone as high as three hundred in one of them. These were the remarkable things in the blood picture. A good many of the cases are rapidly fatal. On the other hand there have been recoveries. The Boston men tell me they have seen patients recover after transfusion. Our first patient came to the hospital when six hours old. The color increased in depth very rapidly. Within about twenty-four hours the child had an enlarged spleen. He seemed very ill. We treated him by transfusion, giving him, all told, seventeen transfusions to keep him alive while we might come to some decision as to other possible therapy. Finally I concluded that we were dealing with a hemolytic anemia, for which the spleen was probably responsible. Splenectomy was decided upon and performed. So far as I know this is the only case in which that has been done. We gave a transfusion before operation, which brought the hemoglobin to about twelve grams. The hemolysis, which had been extreme, stopped immediately after the operation, and the hemoglobin remained at the high level. No more transfusions were given. The deep color of the plasma disappeared almost immediately. That child, so far as we could see, was cured by splenectomy.

DR. GRULEE.—What effect did it have on the nucleated cells?

DR. COOLEY.—They disappeared quickly. The child was then about five weeks old. We had kept it alive that long by the frequent transfusions. What happened subsequently was that, his nutritive state having become low, and the abdominal wall very thin, the splenectomy wound gave way, and the child died of

peritonitis about ten days after the operation; but he had no further signs of the hemolytic process. As to the second one that I saw; while we were getting ready to do the same thing on the second day of life, the baby grew rapidly worse, so fast that we had no chance to operate. We had waited a day because of the information from Boston of recoveries after transfusion, and as this was a vigorous baby we thought we could wait a little for possible improvement, giving a fair-sized transfusion, which seemed to do harm rather than good. At least, the hemolysis was much more intense after it. In another case, I should urge immediate splenectomy.

DR. GRULEE.—We have had no success with transfusion.

DR. COOLEY.—My feeling is that in another case I would take out the spleen as soon as I saw the child, from our result with that one case. There is a very interesting thing that I never thought of until I saw that second patient and watched him die. The deep color is not real jaundice. It is nothing but the color of the blood plasma showing through the skin, and it disappears almost immediately after death. There is no pigmentation of the skin at all. The German literature would lead one to think that nearly all of these children are edematous. That was not true of either of ours.

QUESTION.—What is the relation of this condition to icterus gravis?

DR. COOLEY.—I am not sure that I know what icterus gravis is. Different diseases seem to have been called by that name. One of the common ones, apparently, is a hemolytic septicemia of the newborn. I am inclined to believe that familial icterus gravis and this erythroblastosis are the same thing.

DR. SANFORD.—I happen to have reviewed the literature of icterus gravis. We reported two cases. In all of the reported cases of icterus gravis there was no accurate blood count made, so that we have no way of knowing whether the cases were erythroblastosis. Gregory in 1928, Struthers in 1926, Hart in 1925, and Rosenbaum in 1925 reported cases of icterus gravis in which nucleated erythrocytes were noted, but an exact count was not given. Our two cases came almost together. I think Dr. Grulee saw the first one on the fourth or fifth day. The blood count showed anemia and there were 450,000 erythroblasts. The child was given a transfusion and appeared better. The erythroblasts dropped to about 48,000 and it was given another transfusion. It seemed to improve slightly, but died soon after.

The second case we saw immediately after birth. The peculiar thing was the remarkable yellow color which Dr. Cooley spoke about. The blood count showed anemia, with 150,000 erythroblasts on the first count. The spleen was enlarged but there were no other findings except intense jaundice. We had a good opportunity to study this child. The stools were normal, the icterus index was 75 and the indirect van den Bergh was highly positive. This child was given one transfusion. It had no effect on the erythroblasts and a second was given. The patient died on the fifth day.

We obtained complete autopsies on both of the children. The striking thing was the erythropoiesis existing in the bone marrow, spleen and liver. The microscopic sections appeared as if we were looking at organs which had been unable to adjust themselves to extrauterine life. In other words, we were looking at an intrauterine blood picture. For some reason or other these infants had been unable to change their blood-forming organs from persisting fetal conditions.

DR. COOLEY.—We have recently seen two cases of a thing I had never heard of, and I have found nothing about it in the literature, though I have not made a thorough search. It is what one might call congenital macrocytosis. I think that

would be a fair description. The first patient I was called to see in consultation. I found a child four days old, apparently dying. He had feeble, irregular respiration, with what looked like paroxysms of pain. The obstetrician had discovered a tumor in the right lower quadrant, and we speculated what this might be in relation to the symptoms. We decided to send him to the hospital for investigation, but had to make use of artificial respiration before we could remove him, the respiration having stopped entirely for a time. At the hospital he was given an infusion of saline solution, which revived him considerably. It was decided to investigate the tumor, having in mind a misplaced kidney with kinked ureter, and possible intussusception. While we were preparing for the operation we went over the blood, and found a red count of 7,000,000, with a hemoglobin of more than 28 grams. The tumor was found to be a kidney, placed low down in the pelvis and anchored there, with the ureter coming off the top and dilated. The paroxysms may have been a sort of Dietl's crises, or, as it occurred to me later, they may have been symptoms of asphyxia, as the child was really being suffocated by his own blood. The cell volume was about 75 at first. The high hemoglobin has continued, though it has gone down considerably because the red count has fallen. The present red count is about 3,000,000, with a hemoglobin of 15 grams.

An interesting thing is that the father has the same condition, in a less pronounced form, and the first child in the family died of what seems to have been the same thing. Both father and child have pronounced macrocytosis and a very high color index. The red cells are normal except for the size. The father tells us that his health has always been good, but that his blood pressure is high for his age. The baby is doing perfectly well. Two weeks after this first case we had another. The second one was a premature with just the same picture, very large cells and a tremendously high hemoglobin content. We are giving the same treatment—dilution of the blood with saline solution. We have not bled them except to take considerable amounts of blood for study. Both babies go along like perfectly normal children, with rather low red counts but all the hemoglobin they can possibly use. There are no abnormal cells, and they are very uniform in size. It is simply that these children are making cells that are a great deal bigger than the average.

DR. HAMILL.—They continue large?

DR. COOLEY.—Yes. The father's are of exactly the same type. It was a curious coincidence that when we came to examine the blood of the father and the mother, who were not at all related, we found that both had hemoglobin above the normal. The mother's blood is all right otherwise. She does not have the big cells. She is simply unusually well supplied with red cells and hemoglobin.

Baar and Stransky's book, which is a mine of information, makes no mention of macrocytosis, which was surprising to me, because it is a characteristic of several types of anemia.

I am inclined to think that of the anemias of early life those classified as nutritional anemias are perhaps the most common and most important, at least during the first year. Dr. Sanford has some material on them.

DR. SANFORD.—I am going to bore you for a minute by briefly reviewing some of the history of these nutritional anemias. I think one reason for the difficulty we have with this subject is that some of them are not correctly classified. I am referring to anemias due to a certain blood deficiency. We know that some of the minerals, iron, copper and possibly manganese, enter into this. Bunge did the first work on this in 1889. He found that puppies became rapidly anemic from the second or third week after birth. Studying the livers of these animals, he found that their iron content decreased rapidly after birth. He

studied the embryo also, and found that the greater part of the iron was laid down in the last weeks of fetal life. Hugounenq in 1889 found the same thing to be true of the human fetus. He examined the livers of children who had died from causes other than anemia and found that throughout the first year the iron content of the liver decreased steadily.

In France, the anemia in question had been known for some time. In 1771, Sauvage reported a case of "chlorotic" anemia in a child of eight months. Hallé and Jolly in 1903 reported a number of similar cases.

Czerny, in 1912, upset things by his report on anemias in children which he considered to have nothing to do with iron deficiency, but due entirely to milk injury. Therapeutically, iron had no effect, he said, in either organic or inorganic state, and the only treatment was to take the children off a milk diet and give them a high feeding of cereals, vegetables and meat. This idea of Czerny's colored the literature to 1920.

Studying the etiology of this condition, we always find that the children have been on a restricted diet or have been exclusively milk fed. It does not develop much before the second or third month, and reaches its maximum about the end of the first year. Then, if the child is put on a mixed diet, there is a slow rise in the hemoglobin and the red cells throughout the following year. Other deficiencies than that of iron have been accused of being the cause. All the vitamins have been considered, especially vitamin D. The reason for that is that we frequently find this anemia associated with rickets. I think we will all agree that curing the rickets will have no special effect on the hemoglobin and the red cells. There are other definite factors besides the iron deficiency. First, as to the mother. Czerny was certain that these children were predisposed from birth to anemia and coined the term "constitutional anomaly." As a matter of fact, there is no direct evidence that the mother's condition has anything to do with this kind of anemia. In white rats it is possible, by producing anemia in the mother, to make the brood anemic. On the other hand, Helen Mackay did not succeed in increasing the hemoglobin of the child by feeding iron to the mother.

Predisposing factors are important. First, the age of the child and its weight at birth. The iron in the liver is laid down in the last months of fetal life. If the child is prematurely born, it will lack a certain amount of iron. This will be apparent as the child increases in size. Secondly, the smaller the baby is at birth the more likely it is to become anemic. If we stop to think of that, it is quite obvious, because the small baby gains weight so much more rapidly than the large one. A baby with a birth weight of five and one-half or six pounds will weigh fifteen or sixteen pounds at six months, while the eight-pound baby will weigh little more. The one child has doubled his birth weight while the other has tripled his. Both started out with an iron reserve roughly proportional to the birth weight, but owing to the more rapid growth of the smaller baby his reserve is used more rapidly than that of the larger child.

Season has apparently no effect at all. Sex incidence has nothing to do with it. This anemia is found not so much in the breast-fed child as in the bottle baby, and particularly in the artificially fed child who is kept on an exclusive milk diet throughout the first year.

The blood picture shows a decrease in hemoglobin but not a corresponding decrease in the red cells. We find the hemoglobin around 50 or below, while the red cells stay around 4,500,000. It is unfortunate, as Dr. Cooley has said, that the findings of various investigators are not readily correlated. A great deal of this work has come from England. There they still use the Haldane method, which he published in 1901. According to his calculations the oxygen-combining capacity of the blood is 18.5 c.c., corresponding to 13.8 grams of hemoglobin.

This method will give, in a normal person, a hemoglobin of 115 per cent, which is misleading. That has been pointed out by Haden and Sackett. Most figures on the frequency of this condition give, therefore, a higher value of hemoglobin than we should have.

Helen Mackay in England has done the most comprehensive work on nutritional anemia. She has been working on it for the last five or six years and estimates the frequency of the condition at 40 per cent in breast-fed babies and 70 per cent in artificially fed babies. She arrived at that conclusion in a rather devious way which I think should be mentioned.

By feeding iron to artificially fed babies she was able to get a hemoglobin content in the children of around 80 per cent. Therefore she assumes that this is the normal hemoglobin for the child from three or four months on. We have no more right to say that this is the normal hemoglobin for a child of that age than we have to say that because by the use of parathyroid we can raise the blood calcium to 12.0 or 15.0 mg.; therefore this must be the normal amount. This certainly is not correct. She calls any child anemic whose blood varies 10 per cent from these figures, and if we use her figure we arrive at 40 per cent for the breast fed and 70 per cent for the artificially fed.

In her series of cases, she finds that after the high hemoglobin content at birth we have a rapid fall to about 65 per cent at the second month, a slight increase to around 75 per cent the third and a fall again to around 65 per cent throughout the latter part of the first year. Personally, I don't think a child should be considered anemic if its hemoglobin is 60 or 55. It should be considered in the anemic class only if the hemoglobin is 50 or below.

DR. COOLEY.—I feel much as you do, Dr. Sanford, that it is wrong to say that a perfectly normal child is anemic because his blood does fall, during the first few months of life, below the figure which it will reach in later months.

There is, however, in Helen Mackay's report, one thing which I thought very interesting. In her series of cases, quite a large series, and I judge pretty well controlled, she saw about 50 per cent as much infection in the children who received iron during this period as in those who did not. I thought that was a very good argument for giving children iron at that stage, if the observation of others should substantiate that particular finding. Hers were dispensary patients of the poorer class, children who had not been adequately fed.

Another thing that suggests itself from that report, which I think one might think seriously about, would be the possible desirability of giving iron to all premature babies. That argument, of course, is not hers originally. The proportion of iron reserve to the birth weight of the baby and the relative insufficiency in the baby who gained particularly fast would apply quite obviously to most premature babies, if they are well cared for.

Of course, we do not make blood counts on most of our prematures. In the early weeks we do not do anything that we can help except feed them. I have been wondering, since I read that report, whether, if we should give all our prematures iron, we should have less of the anemia which we take for granted in later months.

DR. McGEE.—Would egg do that?

DR. COOLEY.—I should say that it would have some effect. Whether it would be sufficient I cannot say. Dr. Mackay used iron and ammonium citrate. That brings up the question of the copper content of those preparations.

DR. ALDRICH.—Did they give any material on the relationship to gain in weight? You spoke of the data on infection and I wondered whether she gave any on gain in weight.

DR. COOLEY.—It is quite an extensive report. I do not remember offhand whether her tables show a relationship between iron treatment and gain in weight.

The child who is anemic at the time of its most rapid growth is likely to suffer permanent effects if the anemia is not cared for. I think this is really one of the commonest and most important of the anemias of early life, and the most neglected.

Dr. Mackay added iron to the milk given. They were using an evaporated milk, and she got the manufacturers to incorporate the iron, in the form of the double citrate, in the canned milk. It is a cheap way of managing the administration, and seemed to work well.

DR. HENRY E. UTTER (PROVIDENCE).—During his service at the Providence Lying-In Hospital in 1930 and 1931 Dr. William P. Buffum gave to all premature babies three to five grains of iron and ammonium tartrate in the total day's feeding. It was noticed that the pallor appearing in most premature babies was lessened or did not appear at all.

DR. COOLEY.—I have asked the man in charge of our prematures to take up this question and see what we can find out about it.

DR. HAPP.—May we discuss the relation of infection to these anemias now, or do you want to take that up later?

DR. COOLEY.—If I have the right idea of what you mean, I think we might take it up now. It seems to me that infection is particularly likely to bring to a head, so to speak, the effect of exhaustion of the iron reserve. You may not realize there is anything the matter with one of these children until it gets an acute infection. Then all of a sudden it develops pallor very acutely and the anemia comes strongly into evidence.

DR. SANFORD.—Dr. Mackay said that about 50 per cent of the anemias in her series were due to infection. I was trying to think this afternoon, and cannot remember a single case of nutritional anemia we have had in the hospital in the last three or four years in which there has not been a history of infection.

DR. COOLEY.—It seems to bring the anemia into evidence.

DR. HAPP.—It seems to me, Dr. Cooley, that the physiologic anemia an infant acquires during the first year and which the premature infant is more susceptible to and always manifests in a more marked degree, is aggravated by the infections, and the anemia again aggravates the infections. The more anemic he becomes, the more susceptible he is to infection. The more infection he gets, the more anemia he develops, so that it is a reversible reaction. It seems to me important that in the first year we should always be on the lookout for this anemia, particularly in those infants who have frequent infections, as otitis media, etc. If those infants are given iron, they will do much better.

DR. COOLEY.—I should like now to take up some considerations regarding treatment. First, as to the use of iron and these recent ideas as to the value of adding copper or manganese or both. There is certainly a tendency just now to use very much greater doses of iron than were formerly in vogue. I think that is true in clinics all over the world. Everybody is giving what would formerly have been considered enormous doses, and I believe we are getting better results. Some of us have been a little suspicious as to what may be the reason for that, particularly in the light of this recent work on copper and manganese, and on investigation we have found these metals are constituents of practically all of the iron preparations we use. The value of copper in the anemias of human beings has been a good deal disputed. Josephs showed pretty clearly, I think,

in his paper last year that it was of use. Isaacs is one of those who disputes this, but I doubt whether he has considered how much copper there is in the enormous doses of iron he uses. Even these very large doses do not contain the amount of copper in proportion to the weight of the animal that Hart and Steenbock say is necessary. We have made the calculations in regard to our own dosage, and found that the copper is quite a little less than the theoretical requirement. It is still an open question, then, whether the large doses of iron may be effective because of their copper content.

Another question has come up in my mind. We know that iron needs hydrochloric acid for its absorption. We know also that many of our anemic patients have pretty low gastric acidity; some of them will not have any for considerable periods. Might it not be worth while to go back a little in our therapeutics? When I was a student the favorite iron preparation was the old-fashioned tincture of ferric chloride, and I wonder occasionally whether it was not the most logical one to use. Of course, others are used because of their more ready solubility; a curious contradiction to this is the free use in Germany and in many clinics in this country of the saccharated carbonate, given in solid form. I think one would have to expect that a good deal of that would go through unabsorbed, and perhaps that is another reason for the relatively tremendous doses of that preparation given to children. I do not quite know why we do not go back to ferric chloride, and why we do not do the sensible thing that Dr. Mackay did in incorporating the iron in the babies' feedings.

DR. McGEE.—Dr. Haskell, who taught formerly in Richmond, quite an experimental type of man, said that according to his way of thinking the syrup of the iodide was a good preparation easy to give and quite concentrated.

DR. COOLEY.—That is in the same class with ferric chloride except that there are people who cannot take the amount of iodine that would be given in that way.

DR. SANFORD.—The idea of giving iron medication is to place the iron in such a way that the child can use it. The place that iron must be synthesized from is the liver. The idea is to get that iron into the liver in an assimilable form. When you give large doses of iron such as seven grains a day, if any of you have determined the iron in the feces, you will have found that nearly all of it is going through unabsorbed. The more you give, therefore, the more possibility you have that some of it will reach the liver.

It seems to me that the thing to do is to give the iron in some way that ensures its being utilized without all this wastage. We worked on such a method and finally decided the one most easily used in children was the giving of a colloidal suspension intraperitoneally; the idea being that it is taken up by the lymphatics of the intestine and stored by the liver cells for use. This has been shown by animal experimentation. In giving this, the average injections are two a week for four weeks, making eight in total. We give only eight milligrams of iron for each injection. After the treatment is finished, we have given only sixty-four milligrams instead of the immense amounts given by mouth.

In regard to copper, I might say that iron hydroxide, which is the form we use, contains no copper. I think that if the iron is given in this form in which it can be utilized by the organism no copper is necessary. There is no copper in the hemoglobin molecule and the amount of copper given is so infinitesimal that it must act as a catalyst in the iron metabolism.

In giving iron intraperitoneally, while it is found in the liver cells within a week, we don't get any particular rise in hemoglobin for about a month. It seems to take about that length of time before the blood begins to take up the iron

from the liver. Our experience has been that iron chloride, given by mouth, was harmful to the teeth. That was the reason it was discontinued.

DR. COOLEY.—I have had no recent experience with it. I know that when I was a young practitioner that was one of the things they were troubled with, which should be avoided by Dr. Mackay's method of putting it in the food. One of the things she reported which was quite surprising to me was that she had to cut down the dosage very considerably at the beginning of the administration because of diarrhea and vomiting, so that she had to work up the dose gradually.

DR. SANFORD.—We do not use this form of treatment unless the hemoglobin is forty or below. We make the diagnosis and give the first dose in the hospital, then follow the patients in the out-patient department.

DR. HAPP.—It seems to me the anemia we are discussing in infants is analogous to that produced in rats by a milk diet. The advantage of adding copper or other catalytic agents to iron is that you get the same result with the administration of less iron. So that, if infants during their first year show anemia, it is well to give small amounts of copper in addition to iron, because in that way it is not necessary to give such enormous doses of iron to get the same results.

DR. COOLEY.—I believe that for the ordinary mouth administration the addition of small amounts of copper is desirable. So far as our own results go, we seem to have got along with smaller doses of iron and got more rapid cures. The blood has come up faster.

DR. McGEE.—Both the hemoglobin and the red cells?

DR. COOLEY.—It is the hemoglobin I am thinking of particularly, because that is, after all, your most important criterion.

There is another problem which one meets not infrequently in private practice —that of the patient whose hemoglobin is brought up readily enough to eleven or twelve grams and sticks there, as if the marrow had reached its limit of productivity. Here is where we have felt that we got results from the so-called secondary liver fraction, a by-product from the manufacture of the pernicious anemia extract. One would not use transfusions in such cases, but they are trying sometimes. The children are not really ill, but a bit below par. I think this secondary fraction has been of use to us here.

I have listed for discussion some therapeutic agents of reputed value, such as some of the vitamins, lights of various types, the x-ray. Personally, I should be inclined to skip the vitamins and the lights unless some of you have something particular to say in their favor, because I do not believe they have any important place in the treatment of anemia. I can conceive of special conditions where the destructive action of the x-ray might be put to good use, but I know of no other place for it.

I have seen not a few of these obstinate cases of which I have spoken in which I have found that the best treatment I could prescribe was not drugs at all, but plenty of outdoor air, sunshine and play. This is often remarkably effective in the child that has been pretty much shut in for the winter in our Michigan climate. Another point. In these obstinate cases in which I have not succeeded in getting anywhere with treatment, I have rather too often discovered that my trouble was due to faulty diagnosis; missing something like incipient tuberculosis, a sinus infection, or anything else that would tend to hold down marrow activity. Complete diagnosis is pretty important in these obstinate anemias.

Almost without exception we start treatment of our grave anemias with transfusion. Our reason is this. My conception of what transfusion does is that it brings the hemoglobin quickly up to what one might call a working level. Hemo-

globin is the common carrier for all metabolic processes, which cannot go on normally without oxygen. If the hemoglobin is too low you cannot have normal metabolism. Without normal metabolism you cannot build hemoglobin rapidly. My main reason, therefore, for the use of transfusion, aside from the obvious one of making up blood loss after hemorrhage, is to raise the hemoglobin promptly to a point where the general metabolism can function satisfactorily. Apparently this is more important in acutely developing anemias than in the more chronic ones, in which the metabolism seems to become adjusted gradually to working fairly well at a low level.

In the treatment of a simple iron deficiency anemia, one or two transfusions will often effect a cure if you use no other treatment except to put the patient on a suitable diet. We are satisfied with our theoretic basis for this treatment because it works so well in practice.

DR. SANFORD.—I think Dr. Cooley is correct in his statement that if you have a badly anemic child you have no time to wait three or four weeks for your iron to take effect. Our policy, in all such cases, is to give a transfusion immediately and then begin our iron therapy. If there is absolute iron deficiency the transfusion will not hold the hemoglobin up for more than five or six weeks. After that time there is a definite fall unless some kind of iron therapy is carried out.

DR. COOLEY.—Wouldn't your diet cover that? That has been our experience. I am just getting a series ready to report, in no one of which did we have to give iron; except that when one of the children, some time after leaving the hospital, developed a severe, prolonged otitis we gave iron as a possible prophylaxis. The others had only the iron found in an ordinary good diet. There was no recurrence or falling back.

DR. HILL.—I have had about seventy-five cases over a period of ten years, in which apparently we got remarkable results from fifteen or twenty c.c. of blood given intramuscularly two or three times a week.

DR. COOLEY.—I should not doubt the possibility of that. Similar results have been reported from repeated small intraperitoneal injections. If I can get by with one or two transfusions I would rather do that than use a lot of other types of injections. For one thing, it is easier on the child.

There are some other things to be considered about transfusion. As to the method; I don't know that anything special need be said about that. I find that every clinician has his own pet method. The main thing is to get the blood in. The reactions which sometimes follow transfusion are another matter. We have had a good deal of experience with unpleasant reactions of one kind or another. One thing that has bothered us is the severe hemolytic reaction occasionally encountered when a second or third transfusion has been given from the same donor after an interval of two weeks or more. We have had this happen several times where the first transfusion from this donor had gone without the slightest trouble. These reactions are not ordinarily fatal, but might be if the patient were in very bad condition. We have finally ceased giving second transfusions from the same donor, unless within a week of the first.

DR. HAPP.—We use blood transfusions frequently, not citrated, whole blood from a fasting donor. That is, the donor does not receive any breakfast, and that seems to lessen the number of reactions.

DR. COOLEY.—I don't believe it would prevent this type I am speaking of.

DR. HAPP.—The blood is given slowly, and the transfusions should not be given too closely together. In the anemias we are discussing it is not so neces-

sary to give them close together as in a hemorrhagic disease. Rather than use the same donor twice we use a professional donor.

DR. COOLEY.—That is all right when the people are able to pay a professional donor. Most of our people are not. Sometimes we can get a volunteer from the police or fire department.

DR. McGEE.—I wonder whether those donors had any allergy.

DR. COOLEY.—I think the thing is an anaphylactic phenomenon, but I do not suppose that allergy in the donor has anything to do with it.

DR. SANFORD.—If the transfusions are given intraperitoneally, I don't think you will have trouble. We have not had any reaction like that.

DR. McGEE.—How much do you give?

DR. SANFORD.—We usually regulate it by the size of the child, for the small baby fifty c.c., for the larger one, one hundred.

DR. COOLEY.—Do you feel that your intraperitoneal transfusions are efficient?

DR. SANFORD.—If the child is in poor condition, it will not absorb it as well as if it were in better shape.

DR. COOLEY.—Of course, the kind of transfusion reactions that result from carelessness in technic should not happen. There is another kind that has bothered us. That is the white cell reaction. I think that almost anyone who has made use of transfusion in certain conditions must have seen it followed occasionally by an extreme drop in the white cell count. Sometimes the red cell count falls at the same time. So far as I can find out, there is little information as to the reason for this. It is particularly likely to happen in diseases involving the white cells, such as leucemia and Hodgkin's disease. Doane published one paper on it, describing a method of matching the bloods which we have not been able to follow satisfactorily. We have had several unpleasant experiences with almost complete wiping out of the white cells.

DR. HILL.—Any change in the differential?

DR. COOLEY.—I couldn't tell offhand. Without being able to say definitely, I think the loss was chiefly in the granular cells and platelets. We have learned to be cautious about transfusion in these white cell disorders. It is wise to make the first one small to see what is going to happen.

DR. HILL.—It seems to me there has been an increase in the destructive diseases.

DR. COOLEY.—Don't you think your diagnostic methods are better? While the routine may be the same, you may be a little keener in picking them up.

DR. HILL.—It seems to me we have seen more of them in young children, and I wondered whether anyone else had the same experience.

DR. COOLEY.—I think we differentiate them better than we did, but I don't think we are seeing any more of them.

DR. HAPP.—The Doctor just mentioned the frequency with which one sees peculiar types of anemia associated with leucopenia and decrease in the granular cells. In the past few years, more and more attention has been drawn to that group. In general, those cases have been reported under four classifications. I am bringing them up to ask whether they represent four separate entities or whether they are possibly related. One is the group called agranulocytosis. The second is the group reported collectively as aplastic anemia. The third group is

spoken of as aleucemic leucemia. The fourth is a group called sepsis with leucopenia.

The points which these cases have in common is that they occur in children who have been previously well, without any obvious etiologic factors. The onset is more or less insidious, with the appearance of pallor and fatigue, and generally fever. They are generally progressively fatal in the course of a few weeks or months. The anemia, which is progressive, does not respond to transfusion or to the administration of iron, or to any of the methods beneficial for simple anemias. The characteristic thing is a progressive fall in the number of white cells, sometimes down to a few hundred, and with the fall in white cells a practical disappearance of granular cells in the circulating blood, so that the blood cells are almost entirely lymphocytes.

Bigler and Brennemann reported, in 1930, a group of cases under the heading of "Sepsis with Leucopenia." These cases presented a picture of fever of septic type, anemia, progressive leucopenia and neutropenia, thrombocytopenia, and progressive downward course to death. Petechial hemorrhages were present in a number of the cases. In the nine cases of this group positive blood cultures were obtained in only three. No necrotic lesions in the mouth were found.

Empey and Proescher recently reviewed the literature on the condition known as agranulocytosis, and reported a case. These cases had certain points in common with those reported by Bigler and Brennemann in the progressive course with leucopenia, neutropenia and anemia, but thrombocytopenia and hemorrhagic manifestations were absent, and the blood cultures were sterile. Pain in one or more joints without local redness or swelling was noted in both of these series. Empey and Proescher's case showed in addition necrotic lesions on the mucous membranes, and at autopsy areas of focal necrosis in the bone marrow and liver. The granulocytic elements in the marrow were completely replaced by lymphocytic elements.

During the past few years I have studied several cases presenting a clinical picture similar in many respects to that described in the above mentioned reports, which followed a progressive course to death. At autopsy there was found infiltration in the liver, spleen, bone marrow, glands and kidneys with lymphocytes and aplastic marrow. These cases have been classified as aleucemic leucemia.

These children in the course of their disease, which runs from a few weeks to a few months or a year, show at some time immature white cells in the circulating blood; that is, immature lymphocytes back to lymphoblasts, and immature cells of the granular series back to myeloblasts. In some of the children the number of these immature cells is very small so that from examination of the blood one is unable to make a diagnosis of leucemia because of the paucity of abnormal cells. In others there is a large number of premature, immature cells. Some of the children have hemorrhages and some do not. The cases which are acute and terminate in a few weeks are more likely to show hemorrhages than those that last a long time. The spleen is not much enlarged in these children; it is slightly enlarged, and as the child goes on toward a fatal termination the spleen often gets progressively smaller and disappears from palpation. The same is true of the glands.

Fever is rather a constant finding. One is unable to state positively whether sepsis is present, except that the blood cultures are generally sterile. In other words, the resemblance in the clinical pictures of sepsis with leucopenia and leucemia with leucopenia is so close that the differentiation is not always possible during life. Bigler and Brennemann state (page 529): "Aleukemic leukemia can be easily differentiated, if at some phase there has been a high leucocyte count or if myeloid cells are found in the blood stream." It has been my experience that aleucemic leucemia, so called, may run its entire course without leucocytosis.

The presence of early myeloid or lymphoid cells in the blood stream is a common finding in children with acute infections, as pneumonia and otitis, and may occur in sepsis.

I feel that acute leucemia of the aleucemic or leucopenic type is more common than is generally recognized, and this opinion is expressed by Baar also. Time does not permit a further discussion of this interesting subject. Pathologic examination is necessary to establish definitely the diagnosis, and even then the differentiation may be difficult in view of our present lack of knowledge of the subject.

DR. COOLEY.—Most of the men who write of the blood disorders nowadays speak frequently of having to differentiate something or other from aleucemic leucemia, but they do not seem to make that diagnosis often. I think we see it more commonly now than formerly. Most of us have it in mind all the time, whether we make the diagnosis often or not. It has not been my fortune to see many cases. Dr. Helmholz told me this morning that they had been rather surprised at the number they saw in their clinic, with the diagnosis confirmed by the pathologist. It has been our experience to see a number of cases with a blood picture such as Dr. Happ has described, where there was progressive anemia with not only a very low count of granular cells, but often no granular cells at all, and no platelets.

That particular condition, where there was practically complete agranulocytosis, has been in all our cases in connection with infection, and that infection almost always of bone, such as osteomyelitis, chronic sinusitis, or something of that kind. One case developed in the course of a prolonged enteritis. It was of a less severe type, and recovered. These cases did not have the postmortem findings of leucemia, and we have called them "secondary aplastic anemia." I think that is the best description one could give. We have become convinced that when this condition once develops in severe form the patient does not get over it. We have had several that apparently were recovering, but with any recurrence of infection the anemia came back in as severe form as before. In one case, both the platelets and the polymorphonuclears came back nearly to normal for a time. This had been a bad sinusitis. Then the child developed a mastoid infection, and that finished it.

There is one other disease in which a blood picture of this kind is seen at times, and that is Hodgkin's disease. It is not so persistent a picture, but it has given us a good deal of trouble in diagnosis, and sometimes we have only been able to make it at autopsy.

The allotted time being over, the Round Table was adjourned at this point.

Academy News

Report of the State Committee of Kansas

At the last meeting of the Kansas State Council for Health, Education and Welfare, the following reports were submitted by the various committees of the Council. This state council represents the Kansas Follow-Up Committee of the White House Conference.

Report of Committee on Growth and Development

Further study in the State is needed before the final adoption of normal standards, chiefly as to the determination of a normal range of variability and data concerning the neonatal and adolescent periods. The Committee recommended that a careful and extensive study of a relatively small number of individuals over a

long period of time be undertaken for the advancement of knowledge in this field. Various aspects of the problems of the physiology of growth, endocrine function, nutrition, with the rôles played by vitamins and minerals and the many problems relative to mental growth, were cited as profitable fields for further study. Whatever share Kansas may take in such study, it would seem obvious it be best undertaken through the Medical School of the State University in cooperation with other departments of that institution.

Report of Committee on Public Health Organization

The Committee is of the opinion, from a preliminary investigation of the data already collected and from the experience of the individual members that the medical profession of the State is, and always has been, in favor of public health work, gladly cooperates in the enforcement of health laws and regulations and realizes the value of the educational phase of public health work.

County Health Organization in Kansas: The State of Kansas has 105 counties with a total population of 1,881,000. Ten of the counties with a combined population of over 250,000 not including the cities of Wichita and Topeka, are served by full-time health organizations. Each of the ten full-time county health departments has a physician in charge; five of the counties have in addition to the health officer, one nurse; four have a health officer and two nurses. There is also a full-time office clerk in each county.

The Kansas Tuberculosis and Health Association, the Red Cross Society, life insurance companies and other groups are contributing their whole-hearted support and also financial aid in many communities. The State Chamber of Commerce and similar organizations are assisting materially in the educational phase of public health work.

The following recommendations are offered by this Committee:

1. Recommendation is made that R.S. 72-5201 commonly referred to as the Dental Inspection Act be amended so as to state specifically who is to pay for the inspections; to state specifically what local officer or officers are responsible for the administration of the Act, and that the Act be further amended by the addition of a penal section, providing a penalty for the failure or misfeasance of the officers designated for the administration of the Act.
2. That a Dental Division be created in the State Board of Health.
3. That facilities for hospitalization of tuberculous individuals be increased.
4. That all unofficial health agencies, especially in large cities or localities having a large population, engage or designate a common executive secretary for the purpose of correlating their health activities.
5. That all boards, having for their purpose the protection of the public health of the State, be combined so far as possible under one board or agency and especially for the purpose of inspection.
6. That full-time health service be given all communities in the State of Kansas.
7. That all county, city and school nurses make monthly reports to the State Board of Health in order that the State may receive due credit for health work done.
8. That a satisfactory method of operation for the Crippled Children's Law be developed soon.

Report of Committee on Medical Care of Children

Medical Education in the State: The State University has increased the hours of pediatric teaching to 157, approximately 3 per cent of the total number of hours given in the Medical School. This is an increase of over one-half as compared with three years ago. With the increase in pediatric teaching hours, the staff has been increased and various clinics have been established.

Health Education: Due to the fact that Kansas has so few pediatricians, no worthwhile report in regard to regular health examinations made on children in private practice can be given.

There are many organizations, city, school, parent-teacher associations and other groups throughout the State sponsoring regular health examinations during the past few years, some supervised by physicians, others by city or county nurses. These have been promoting health education to a considerable degree.

In 1927-28, 32,548 children were examined in ten counties, consisting of the pre-school and school groups and in 1930-31, 34,230. The proportion of defective children is decreasing where regular examination programs are carried on. Furthermore, we have determined the fact that of all these children examined in the past four years in the ten counties, 2000 of the average 30,000 examined each year showed defective nutrition, the major defect noted in the reports.

Vaccination Against Smallpox: Eighty per cent of the school children are unvaccinated unless there has been an epidemic of the disease in the community in recent years.

Diphtheria Immunization: There has been a 75 per cent reduction in cases of diphtheria and a 68 per cent reduction in the number of deaths as compared to the previous 5 years. This is the result of 300,000 children receiving immunization against diphtheria. This feature of the report is very gratifying. However, we believe by more organized effort in the future, such as will be arranged, there is room for further education as to the value of this procedure.

Dentistry and Oral Hygiene: The Dental Inspection Law of the State is far from being "air-tight" and until such legislation is provided to make inspection compulsory with penalty provided for failure to comply with the law, or to place such work under direct control of the State Board of Health, we cannot expect best results. It is interesting to note that in the past four years out of approximately 200,000 children examined in ten counties, nearly 50 per cent showed defective teeth. This also should bring to us the thought of how essential it is to have correct legislation along this line of medical care for the children of Kansas.

Mortality Statistics and Birth Rate: Since 1914, we find the ten outstanding causes of infant mortality are: (1) premature birth; (2) congenital debility; (3) diarrhea and enteritis; (4) bronchopneumonia; (5) congenital malformations; (6) whooping cough; (7) convulsions (cause unknown); (8) lobar pneumonia; (9) meningococcus meningitis and (10) bronchitis. To show just what progress has been made relative to preventive measures and treatment, the number of deaths in 1914 of the ten principal causes may be compared with the number of deaths in 1930.

Premature birth	580	505
Congenital debility	561	75
Diarrhea and enteritis	444	164
Bronchopneumonia	230	138
Congenital malformations	177	217
Whooping cough	100	32
Convulsions	56	10
Lobar pneumonia	38	55
Meningitis	35	4
Bronchitis	26	11

It is concluded from this comparison that more emphasis should be placed on instruction in prenatal care, that there has been commendable progress made in reference to advice on nutrition, that the treatment of acute intestinal disorders has been greatly improved and finally, that progress has been made in the prevention

and treatment of the contagious diseases. Thus, the chief preventable causes of infant mortality have been materially decreased since 1914.

Since the last report of this Committee, we have attempted by means of publicity and education to further interest and influence leaders among the 105 counties of the State, in the importance of organization of separate county councils to carry out an extensive study formulated as nearly as possible to the organization of the White House Conference and our State Council.

We have succeeded with the untiring assistance of the State Board of Health in organizing twenty-one councils throughout the State. Each one of these county councils is formulating plans for future work. We believe there should be county councils throughout the State before much headway can be made. After such complete organization, our aim is to gather definite material to be studied and worked out; to establish a state-wide county health organization with well-trained, full-time personnel. The Committee is of the opinion that such county organizations will not only stimulate plans for the medical protection of our children, but also aid the Education and Welfare units of the State Council to concentrate their efforts in a more conclusive manner. The organization of 25 such councils—some with and some without full-time units, already formed in the past six months, serves as a very fine nucleus for a state-wide full-time health program.

Report of Committee on Prenatal and Maternal Care

The number of midwives in the State was not thought to be sufficiently large and this subject was, therefore, not studied in detail. Obstetric nursing is being improved in several sections of the State, but not up to the basic requirements set forth by the White House Conference.

Medical Education: Undergraduate medical training is offered at only one place in the State—The University of Kansas Medical School at Kansas City. Whereas the White House Conference recommended each student shall have participated in 25 confinement cases before graduation, the Committee finds each student in our State University Medical School participates in about 15 cases. It is, therefore, urged that the clinical facilities in obstetrics be materially increased in our State Medical School.

Postgraduate medical training in Kansas is above the average of most states in certain other respects. The Extension Division of the University has, for a number of years, provided short courses and "refresher" courses for practitioners and in this respect has made Kansas a leader among the states. Since the State Board of Medical Examiners in 17 states require more adequate hospital interneship, including an adequate maternity service wherein the young physician shall have delivered some 100 women before he is licensed to practice, the Committee urgently recommends to the Kansas State Board of Medical Registration and Examination, consideration and adoption of this requirement.

Abortions: We have no recommendations to lessen the practice of abortion. We believe more may be done through continued education of our young people than in any other way. It is recommended that every couple receive along with their marriage certificate a small pamphlet dealing briefly and plainly with facts relating to the institution of marriage and the home. In this the question of abortion could be discussed and also proper care during pregnancy emphasized. In our opinion the majority of young married couples would read such a book.

Infant and Maternal Deaths: The birth rate has a rather steady decrease in the last five years and was the lowest in 1931. The maternal death rate was the lowest in 1931 of any of the past five years. There is a steady decrease in the listing "Congenital Debility." Part of this decrease is, no doubt, due to a better classification and improved diagnosis. Premature births are definitely decreasing.

This is encouraging because it no doubt means better prenatal observation. Injuries at birth are showing an increased rate but there is no doubt at least part of this increase is due to a better classification and a more truthful report on the part of the attending physician. It is surprising to note the mortality rates are higher in the cities where better facilities are available than in the rural districts. However, one must realize many cases of eclampsia and puerperal septicemia, as well as other serious conditions are sent to the hospitals in cities after the conditions have developed. When these cases die in the cities they are so reported and the mortality rates are increased.

Publicity: This subdivision of the Committee on Prenatal and Maternal Care was delegated to ascertain the amount of instruction which women of Kansas are receiving in regard to prenatal and maternal care and the best methods of disseminating this information. It is the concensus among the various health chairmen, county nurses, members of the State Board of Health and other interested agencies that very little is being done along this line and there is an urgent need for this type of education in Kansas. In a group of reports by county nurses only six made reference to conferences pertaining to prenatal and maternal care. In counties where a county nurse is employed, it would appear the distribution of literature and the dissemination of information concerning this phase of the work might be handled through that office. However, this could be augmented by workers in civic clubs, the commissioner of the poor, school nurses and the various social service agencies.

Report of Committee on Communicable Disease Control

This Committee has concluded from its surveys that greater efficiency could be secured through:

1. Improved and earlier reporting of cases and carriers.
2. A more general knowledge of scientific preventive measures.
3. Greater use of the facilities now available for the control of communicable diseases.
4. The development of full-time county health departments in greater numbers.
5. Improved coordination between health departments and all agencies that have to do with public welfare.

Report of Committee on Milk Production and Control

This Committee wishes to emphasize the fact that health in the individuals who handle the cows and the milk is of the utmost importance and also that this Committee will advocate and work for regulations that will require examinations of all farm and factory workers who in any way come in contact with the cows, the dairy utensils, or the milk itself. The most rigid rules should apply to workers in the dairy. Market milk inspection in Kansas has been carried out by local inspectors of the various cities where a milk ordinance is in operation. As a part of their regular duties, the inspector of the State Board of Health and those of the State Board of Agriculture in the Dairy Division have made special surveys to determine what degree of service is being rendered by local inspectors and to assist them in establishing new ordinances and efficient methods of doing their inspection work. For three years a special market milk department of the Dairy Commissioner's service has been conducted and in this work the State Board of Health and the Food and Drug Inspectors have cooperated. Timely and extended surveys have resulted in new ordinances being passed, new methods employed and a cleaner, better milk furnished to consumers. Satisfied dairy farmers testify to the value of dairy inspection.

Meeting of Regional Board for Region II

There will be a meeting of the Regional Board of the American Academy of Pediatrics for Region II at 3:00 P.M., November 15, 1932, at the Tutwiler Hotel, Birmingham, Alabama, for the purpose of discussing the policy of this Region for the next year. There will be a joint meeting in the evening also of all members of the Academy and State Health Commissioners attending the Southern Medical Association at 6:30 P.M. at the same hotel. Any physicians who are interested in any phase of pediatrics are invited to attend. These meetings will be held under the direction of Dr. E. C. Mitchell of Memphis.

Meeting of State Chairmen, Region III, Held in Chicago, October 1, 1932

A general discussion was held concerning the best way of carrying through the White House Conference program in each state. The State Chairmen first reported on particular procedures that had been followed toward developing an interest in this program.

Hempelmann (Missouri).—An effort was made to interest the state governor in the conference program. The governor thought the state was doing enough. Later, after a conference with the State Director of Child Welfare, contact was established between the State Director and the Academy of Pediatrics. The Director of Child Welfare is favorable to such cooperation. The mortality rate has been high and a child welfare program is necessary. The impression prevails that progess is being made.

Cooley (Michigan).—Child welfare work done by the Children's Fund of Michigan enables the state to carry through a very good welfare program. Postgraduate teaching and a lot of other activities are taking place. The Director of the Fund is a member of the Academy. The Children's Fund is doing a good job. State activities of this nature are better done by properly organized health departments than by leaving the program of preventive medicine to private practitioners. It is also believed that the Academy should notify the state authorities that Academy members stand ready to do what they can, as in individual tie-ups with the state program. Activities by these men as individuals would probably be more effective than having the society act as a unit.

Torian (Indiana).—Organization of the Pediatric Department of the University of Indiana is now proceeding on standards set by a subcommittee of the conference. Effort was also being made to organize postgraduate work in the state. The milk situation is being improved. No other work has been undertaken, more for want of knowing how to go at it.

Carson (Kansas).—An attempt is being made to institute a general immunization program in the state. Carson spoke of the Crippled Children's Committee which was well organized and which at first incurred protests of the practicing physicians. This protest has disappeared as it has been recognized that the program of the Crippled Children's Committee applies to the indigent only. It is hoped that through this organization the program of the White House Conference may be carried through.

Burhans (Ohio).—An attempt is being made to have a committee of the White House Conference act as a subcommittee of the State Medical Society. Dr. Burhans expressed the feeling that the Academy do something toward taking a definite stand on disputed medical practices, for instance, immunization for scarlet fever, and so forth.

Schwartz (Wisconsin).—A report was made of the representation of the Academy membership in the program of the State Board of Control. This Board, working under state jurisdiction, is engaged in redefining minimum standards for child-caring institutions. The Academy membership is taking active part in this very important work. It is hoped that out of this will develop a program of follow-up on the White House Conference.

Stewart (Minnesota).—The activity in Minnesota has related itself in the main to increasing the membership. Uncertainty prevails as to methods of procedure for Academy activities.

Dr. Stewart having succeeded Dr. Helmholz as State Chairman requested that Dr. Helmholz comment also on the situation in Minnesota.

Helmholz (Minnesota).—The Secretary of the State Medical Society is also Secretary of the Minnesota Public Health Association, which does very good work. Dr. Helmholz expressed the opinion that publicity programs for child welfare activities should be put into action through the Medical Society. It is important that pediatricians take an active interest in State Society programs in order to carry through important child welfare activities.

Moore (Nebraska).—The program thus far has included:

1. A survey of pediatric activity.
2. Pressure resulting in putting a stop to a commercial baby show.
3. White House Conference Committee activity. This committee, unofficially appointed by the President of the State Society, is attempting to obtain medical cooperation throughout the state.

Gengenbach (Colorado).—This state is fortunate in having a Colorado Council of State Wide Agencies, composed of all organizations through the state with medical and social-medical interests. Through this organization a committee was appointed to see the Governor, who acted as Honorary Chairman. Dr. Gengenbach was named Director of White House Conference Follow-Up Committee. An attempt was made first to sell the idea to the profession, the Director appearing before various organizations. Cooperation of pediatricians was asked. A State Conference was held on lines of the National Conference in January of this year. Five hundred attended. A Follow-Up Committee has been formed, composed of Section Chairmen. They have also had County White House Conferences to bring the ideas closer to people.

Hess (Illinois).—The Superintendent of Schools, Chicago, was named Head of Conference by the Governor of the State. Through the Head of the Medical Section, Dr. Hess' aid in conference was secured. The conference actually represented four or five counties, including Cook County, not the entire State. No activities have taken place since conference meeting.

Moore (Iowa).—The State Society was asked for a committee on Child Welfare and Protection. An attempt is being made to—

1. Make the State child conscious.
2. Establish medical leadership in organizations having medical problems.

A State White House Conference has been held. One thousand people attended. The proceedings of this conference will soon appear. The Academy members are also taking part in local programs of health education and immunization.

News and Notes

The Central States Pediatric Society at its meeting in Kansas City on October 7 and 8 voted that since each member of the Society is invited to attend the regional clinical meeting of the American Academy of Pediatrics in September, 1933, it would hold no separate clinical meeting. The Society voted, however, to hold its annual business meeting in Chicago at the time of the regional clinical meeting of the Academy at which time the future policy of the Society will be voted upon.

The following officers were elected for the coming year: President, Dr. J. H. Hess, Chicago; Vice-President, Dr. Edgar P. Copeland, Washington, D. C.; Secretary-Treasurer, Dr. A. H. Parmelee, Chicago; new member of the Executive Committee, Dr. Frank C. Neff, Kansas City; Editor, Dr. Heyworth N. Sanford, Chicago.

Obstetrical Institutes were sponsored by the New Hampshire State Board of Health, Division of Maternity, Infancy and Child Hygiene and the County Medical Societies, and in cooperation with the United States Department of Labor, Children's Bureau. The Institutes constituted five lectures and thirteen reels of pictures on the principles and practices of obstetrics. The lecture subjects were:

Mechanism and Management of Normal Labor.
Puerperal Infections.
Occiput Posterior Positions.
Versions—Breech Presentations.
Hemorrhages of Pregnancy—Abortion—Placenta Previa.
Accidental Separation of the Placenta.

The lectures were given by Dr. James McCord, Consultant in obstetrics, U. S. Children's Bureau and Active Professor of Obstetrics, Emory University, Atlanta, Georgia. The courses were held in Strafford, Rockingham, Merrimack, Hillsboro, Grafton, Sullivan, Belknap and Coos Counties. Seventy communities were represented. Doctors were present from Vermont, New York and Massachusetts. Five hundred and seventy-three nurses registered for the lectures. Institute meetings in general were well attended. Approximately two hundred and twenty-four doctors registered with a total of five hundred and twenty visits. Seventy doctors took the complete course. Much favorable comment was received from physicians and nurses in every part of the State, indication being that educational material of this type is very acceptable.

American authors who would like to present papers at the meeting of the **Fourth International Congress** which will meet in London, July 22, 1933, should submit as soon as possible, abstracts of such papers to Dr. Henry F. Helmholz, Rochester, Minnesota. Dr. Helmholz is the chairman of the American committee.

Comments

WE TAKE pleasure in publishing the address of Dr. Cary, President of the American Medical Association. Although it was published in the Iowa State Medical Journal in September, and is reprinted by their kind permission, the Editorial Board felt that it was so important in its concept of the function of the pediatrician, and even more so in its plea for cooperation between the physician and health agencies, that it should have a wider circulation than is possible in a state journal. The lay interest in public health is one that chiefly involves the field of childhood and consequently affects the pediatrician. A discussion such as this, coming from the president of our largest national medical association, is most interesting and important.

IN THE present widespread era of depression our hospitals and children's institutions are faced with a serious situation. Not only will it be necessary to divert the large part of charity gifts to direct relief work this coming year, but there seems to be no doubt that income from endowment and annual gifts, as well as estate gifts for endowment and unrestricted use, will be markedly reduced for several years to come. Boards of Managers throughout the country must face this problem and many are struggling with it at the present moment. We cannot agree with a view we heard recently from a member of a hospital staff that it was a "job for the superintendent and managers." The situation involves everyone, and because of the place of pediatrics in health activities and institutions that have to do with children, the situation calls for the best pediatric thought obtainable.

The great progress of recent years in social development has been largely in the field of childhood. Unfortunately when a financial retrenchment has to be made there is a tendency to cut off the latest or newest and to keep the old. This is not an intelligent attitude, although it is probably the easiest. The intelligent way is to separate the essential from the unessential, the necessary from what may be desirable but can be eliminated without serious loss. Thus we reach the point that a technical knowledge of the problems of childhood must be utilized in separating the essential from the unessential, and so far as the health problems of children are concerned, the pediatrician must assume part of the responsibility of sitting in judgment.

The first great need is to look at the problems of child health and welfare as a whole. Most of us have some particular field or activity we have been interested in and quite properly so. At first thought this seems the most essential and it is apt to hold a disproportionate place in our minds when the subject of retrenchment comes up. The individual whose activities are centered about a hospital for children is apt to give little regard to play activities; the worker who is interested in healthy active children developing social and group consciousness and in the prevention of delinquency, thinks little of diphtheria prevention; the public health physician sees chiefly lowered mortality and morbidity rates, and so on. Each is willing to admit the necessity of retrenchment at the present time, but he feels his own work is so important that the cut should be made along some other line. A budget committee of ten Solomons dividing up a "Community Fund" will not be able to make a single agency happy in this coming year.

It is probably true that every institution or agency is doing some worthwhile or essential work that should not be given up. We must not lose the gain and

progress of recent years. As we see it the solution lies in every agency making an honest fearless examination of its own work to reduce expenses by a careful stock-taking of what is essential and what can be, temporarily at least, eliminated. In this the fact that a job has been created which some one holds should not be taken into consideration, nor the job-holder be allowed to influence the analysis.

Regardless of whether we like it or not for the next few years there will be a decided decrease in the income of every type of agency and institution which has to do with children. Expenses must be reduced, but we must not lose all or a large part of what has been gained through the unintelligent slashing off of activities on a dollar and cents basis alone. Every hospital chief, every medical head of a child caring institution, every pediatrician connected with a public health agency, every physician on a "Board of Directors" should study his individual problem and work so that he can offer intelligent, well thought out ways of cutting expenses without doing away with essential work. It may even be that a great deal of our medical and social work with children has been built on rather insecure foundations, and a period of financial distress will play an important rôle in forcing us to learn to distinguish real values and stress the essential.

THIS issue contains the first of a series of three articles by Dr. Esther L. Richards, Associate Professor of Psychiatry at the Johns Hopkins Hospital. In the October issue her recent book on the "Behavior Aspects of Child Conduct" was reviewed, and we are glad to note in glancing over other reviews that the Journal was not alone in praising and recommending the book. In the December issue an article on the "Neuropathic Constitution" will appear which will be followed in January by "What Is Dementia Precox?"

The Journal of Pediatrics

CONTENTS FOR NOVEMBER, 1932

Original Communications

Child Health and Protection From the Doctor's Viewpoint. E. H. Cary, M.D., Dallas, Texas ---- 537

Parathyroidectomy in Generalized Osteitis Fibrosa Cystica. John Fitch Landon, M.D., New York, N. Y. ---- 544

Immunization to Scarlet Fever by the Inunction Method. Edgar E. Martmer, M.D., Detroit, Mich. ---- 555

A Practical Approach to the Mental Health of Childhood With Special Reference to the Intelligence Aspects. Esther L. Richards, M.D., Baltimore, Md. ---- 558

A Definite Sign Pathognomonic of Paranasal Sinusitis. Maud Loeber, M.D., A. J. McComiskey, M.D., and W. F. Henderson, M.D., New Orleans, La. 565

Growth Problems and Their Relation to Zones of Normal Physiologic Reactions. William Palmer Lucas, M.D., Helen Brenton Pryor, M.D., Crawford Best, M.D., Saxton T. Pope, M.D., and Marion C. Henderson, A.B., San Francisco, Calif. ---- 572

Lobar Pneumonia in Infants and Young Children. Clifford G. Grulee, M.D., and Philip Mulherin, M.D., Chicago, Ill. ---- 593

Temperature and Precipitation in Relation to the Morbidity Rates of Poliomyelitis. John A. Toomey, M.D., and Myron H. August, M.D., Cleveland, Ohio. ---- 601

Intrauterine Pneumonia With Evidence of Healing. Edward Dyer Anderson, M.D., and John F. Pohl, M.D., Minneapolis, Minn. ---- 608

Antiques of Pediatric Interest. T. G. H. Drake, M.B., F.R.C.P.(C), Toronto, Canada ---- 612

Critical Review

Neurologic Conditions in Infants and Children. R. Cannon Eley, M.D., Boston 614

American Academy of Pediatrics

Proceedings Second Annual Meeting of the American Academy of Pediatrics... 635

Academy News ---- 651

News and Notes

News and Notes ---- 658

Comments

Comments ---- 659

What the "DEAN *of* AMERICAN MEDICINE" *says . . .*

Dr. Wm. H. Welch, of Johns Hopkins University, considered by a host of medical authorities "The Dean of American Medicine,"* recently stated at the New York Academy of Medicine:

"Too great economy, as far as health is concerned, because of the current depression, is particularly dangerous to the welfare of growing children. Undernourishment of children, for example, is not likely to show itself immediately, but is bound to show itself later when it is probably too late to remedy it. The ground lost by undernourishment in childhood may never be regained."

Physicians everywhere are being told by patients, that because of reduced incomes their family budgets are being slashed. This means reduced diets. It is well to point out in such instances that, in spite of reduced family budgets, the health of growing children must be safeguarded. Often a child needs only the addition of a balanced, prophylactic compound—such as Maltine With Cod Liver Oil—to render an otherwise insufficient diet completely adequate.*

In conditions due to deficiency of Vitamins A, B (b_1), G (b_2), or D, Maltine With Cod Liver Oil will prove its worth. It is composed of 70% Maltine, a concentrated extract of the nourishing elements of malted barley, wheat and oats—rich in Vitamins B and G—and 30% pure vitamin-tested cod liver oil of high potency in Vitamins A and D. Taken with orange or tomato juice, Vitamin C is added.

Maltine With Cod Liver Oil is biologically standardized and guaranteed to contain four vitamins, A, B (b_1), G (b_2), and D. Biological report on request. The Maltine Company, *Est.* 1875, 30 Vesey Street, New York, N. Y.

Maltine

WITH COD LIVER OIL

ACCEPTED AMERICAN MEDICAL ASSN.

* Taken from Medical Journal and Record

SEE Certifoods ADVERTISEMENT ON PAGE 9